AUSCULTATION AND PERCUSSION.

AUSCULTA, ET COMPONE MEIS SERMONIBUS ORA.

T. WATSON.

AUSCULTATION AND PERCUSSION:

TOGETHER WITH

THE OTHER METHODS OF PHYSICAL EXAMINATION OF THE CHEST.

BY

SAMUEL GEE, M.D.,

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS, LONDON;
ASSISTANT PHYSICIAN TO SAINT BARTHOLOMEW'S HOSPITAL; AND TO THE HOSPITAL FOR SICK CHILDREN.

LONDON:
JAMES WALTON,
BOOKSELLER AND PUBLISHER TO UNIVERSITY COLLEGE,
137, GOWER STREET.
1870.

LONDON:
BRADBURY, EVANS, AND CO., PRINTERS, WHITEFRIARS.

PREFACE.

I HOPE that the result of my labours will prove serviceable to those who wish to master the art of physical examination of the chest. The book may seem small; but I have not spared my pains to make it as complete and clear as possible: what uncertainty appears here and there is not infrequently due to imperfection in the knowledge itself. In the Second Part I have omitted a few subjects which it would have been unprofitable to have treated from the physical-sign point of view alone.

LONDON,
September 9th, 1870.

TABLE OF CONTENTS.

PART THE SECOND.

PHYSICAL EXAMINATION OF THE CHEST.

INTRODUCTORY.

PHYSICAL EXAMINATION AND PHYSICAL SIGNS.

THOSE properties of matter, which are recognised by the science of physics, constitute the objects of Physical Examination. Most parts of the body may undergo a physical examination; the immediate results thereby obtained are called Physical Signs.

The present book is devoted to an exposition of the methods and results of physical examination of the organs contained in the thorax; namely, of the lungs and pleuræ, the heart and pericardium, and the mediastinum, including the large bloodvessels.

The First Part treats of the physical signs considered in the abstract; the pure science of the physical signs.

B

The Second Part treats of the physical signs considered in their subservience to the discovery of disease; the applied science of the physical signs.

PART THE FIRST.

CHAPTER I.

METHOD OF EXAMINATION.

SUPPOSE a patient with the chest exposed, ready to undergo a physical examination: the physician first of all carefully surveys the chest with his eye, this is Inspection: next, with his hand, this is Palpation: he next strikes the chest, Percussion: and lastly he puts his ear to the chest, Auscultation.

Whenever convenient, the patient should remove all clothes from the upper part of the body down to the waist, and stand opposite to the physician. Needful deviations from this rule will be suggested by the good sense of the examiner at the proper time and place. Children who are not able to stand strongly should be stripped naked and held in the arms of a

nurse. In no respect is the physical examination of children more difficult than that of older persons: and the method in all cases should be the same.

CHAPTER II.

INSPECTION.

SECTION I.—SHAPE OF THE CHEST AT REST :

Art. I. Shape in Health :

¶ I. The typical shape.

¶ II. Sub-typical shapes :

i. Alar chest.

ii. Flat chest.

iii. Transversely constricted chest.

iv. Pigeon chest.

v. Rickety chest.

Art. II. Shape in Disease :

¶ I. Bilateral changes in shape :

i. Enlargement.

ii. Diminution.

¶ II. Unilateral changes in shape :

i. Enlargement.

ii. Diminution.

¶ III. Local changes in shape :

i. Bulging.

ii. Shrinking.

SECTION II.—MOVEMENTS OF THE CHEST :

Art. I. Movements of Respiration :

¶ I. In Health.

¶ II. In Disease :

i. Inspiratory Dyspnœa.

INSPECTION discovers the shape of the chest. First; the shape such as it is when the thorax is at perfect rest; that is to say, at the end of an ordinary expiration and during the diastole of the heart. Secondly; the ceaseless temporary changes in shape which the chest undergoes during life, in consequence of the respiratory and circulatory movements.

SECTION I.

SHAPE OF THE CHEST AT REST.

A transverse section of the chest upon a horizontal plane approaches to the figure of an ellipse; between the long and short axes of

which (that is to say, between the breadth and depth of the chest) there is a certain proportion in length. A knowledge of this proportion is the key to a knowledge of the shape of the chest in health, and the unilateral and bilateral changes which that shape undergoes in disease. Changes in the length or height of the chest from above downwards, changes in the direction of the ribs, in the width of the intercostal spaces, in the size of the costal angle, in the arching of the spine and sternum, in the height of the shoulders, and in the projection of the shoulder blades; all these follow changes in the shape of the horizontal ellipse, as the shadow follows the substance.

When the axes of the ellipse are nearly equal in length, and the horizontal plane is nearly circular in outline, the chest is short from above downwards (the floating ribs being excluded from consideration), the ribs approach the horizontal, the intercostal spaces in front are narrow, the costal angle is obtuse or open, the sternum is arched, the shoulders are high, and the scapulæ lie flat on the ribs. On the other hand, in proportion as the transverse axis of the ellipse exceeds in length the anteroposterior, the chest becomes long, the ribs slope downwards,

the intercostal spaces in front are wide, the costal angle is acute, the sternum is straight, the shoulders are low, and the corners of the shoulder-blades project from the ribs. The former is the chest of inspiration or expansion, the latter is the chest of expiration or contraction.[1]

Article I.—Shape in Health.

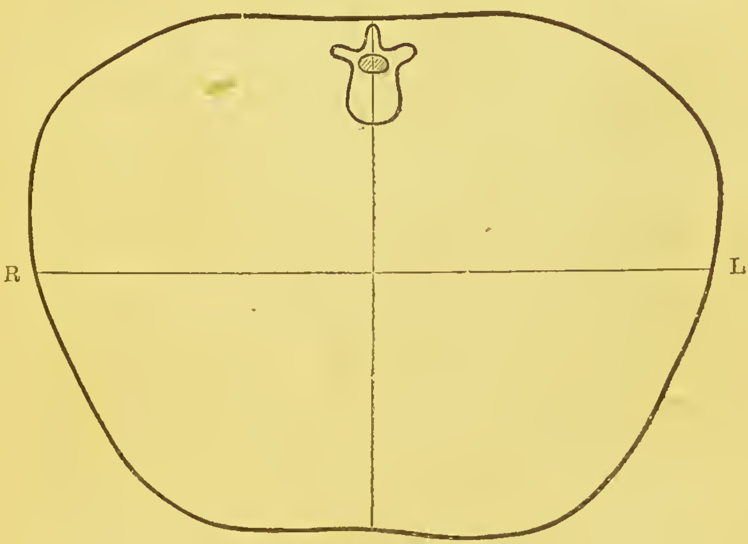
It is easy to conceive an idea or a type of what the perfect human chest should be. But the ideal shape is rarely realised: deviations from the type are present in nearly all persons, who nevertheless may be in perfect health. The more important of these deviations I shall describe under the title of sub-typical.

[1] "Circa hos motus (scil. respirationis) divini Conditoris mechanicen, ad regulas mathematicas plane adaptatam, satis admirari non possumus; siquidem nulla alia in re manifestius ὁ Θεὸς γεωμετρεῖν videatur. Quippe cum pectoris tum coarctatio a quibusdam musculis (quorum munus unicum est contrahere) perfici debeat; res ita instituitur, ut costæ, quæ thoracis, velut parallelogrammi oblongi versus Cylindrum in figuram modo quadratam, cum angulis rectis, pro pectoris ampliatione, modo in Rhomboeidem, cum angulis acutis, pro ejusdem contractione, ducantur." Th. Willis: Pharmaceutice rationalis. 1674, II. 1°. i.

¶ I.—THE TYPICAL SHAPE.

In new-born children the axes of the ellipse are almost equal; the thorax is nearly as deep as it is broad, and is not far from circular. As growth proceeds the breadth of the chest increases more quickly than the depth; so that,

Fig. 1.



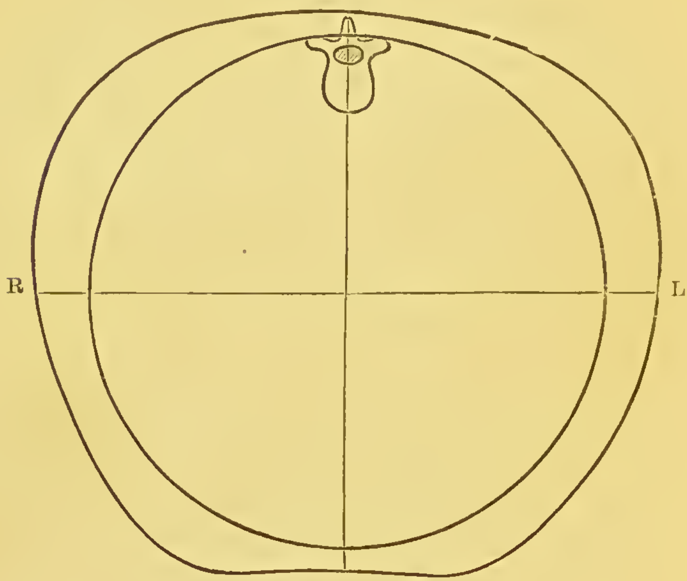
Circumference = 89 centimeters.

Transverse section of healthy adult chest upon level of sternoxiphoid articulation.

by the time the child has cut his milk teeth, a strongly elliptical shape is established. The disproportion between the axes becomes greater and greater (but with diminishing rate of yearly

increase) until the body is fully developed. After which period no further changes occur, until those morbid conditions, which are the almost necessary companions of old age, begin, in most persons, to alter the natural shape, and to make the chest acquire, in the second childhood, much the same figure that it had in the first.

Fig. 2.



Circumference = 40·5 centimeters.

Transverse section of chest of an infant aged 9 months.
(A circle drawn within the tracing for the sake of comparison.)

The Cyrtometer is an instrument by means of which the shape of the chest may be exactly

ascertained and registered. Originated, both in notion and in name, by Andry and Bouillaud, it was by Woillez that the cyrtometer was first made really useful in physical diagnosis. The cyrtometer of Woillez[1] consists of a number of small pieces of whalebone rivetted together so as to form two jointed girths, which may be accurately applied to the two sides of the chest, and which are easily fastened and unfastened before and behind by a simple arrangement. A cheap and perfect cyrtometer may be made by two pieces of composition gas-pipe, drawn out to a diameter of the eighth of an inch, and united by a piece of caoutchouc tubing. The advantage of the composition alloy is that it possesses no resiliency. A few experiments with the cyrtometer are all that is necessary in order to become skilful in its use. The instrument, after having been accurately applied to a given circumference of the chest, is removed, and will then afford an exact tracing of that circumference. It is needless to add that the cyrtometer is the best means of measuring the chest.[2]

[1] Gazette des Hôpitaux, 1857, pp. 134, 193, 205.

[2] I have not described the chest-measurer and the stethometer because of their small practical utility.

In order to illustrate the statements just made respecting the different shapes of the thorax at different ages, I will give some actual measurements, taken upon the level of the sternoxiphoid joint, and so calculated that the circumference always = 100.

Age.	Actual circumference.	Ratio of diameters to circumference.	
		Antero-posterior.	Transverse.
3 months ...	$14\frac{3}{4}$ inches (37·5c.)	26	29
2 years	18 ,, (45·75c.)......	26	32
34 years	$29\frac{3}{5}$,, (75c.)	26	35
48 years	35 ,, (89c.)	27	31

The chests measured being all perfectly healthy, saving the last which was emphysematous.

The semicircumference of the right half of the chest is usually a trifle greater than that of the left; the difference being from a quarter of an inch to an inch. The nipples are seated on the fourth ribs or the fourth interspaces. Where the manubrium joins the body of the sternum there is often a well-marked angle, angulus Ludovici, level with the second rib.

It is convenient to regard the chest as mapped out by certain vertical lines, whereby we can indicate the exact longitudinal situation of any physical sign. The following vertical lines will

be found sufficient: the midsternal, right or left side-sternal, parasternal (i. e. midway between the side-sternal and nipple lines), nipple, mid-axillary, scapular (i. e. the angle), and the vertebral groove. The horizontal level or latitude is indicated by reference to the clavicles, ribs, intercostal spaces, nipples, and sternoxiphoid articulation.

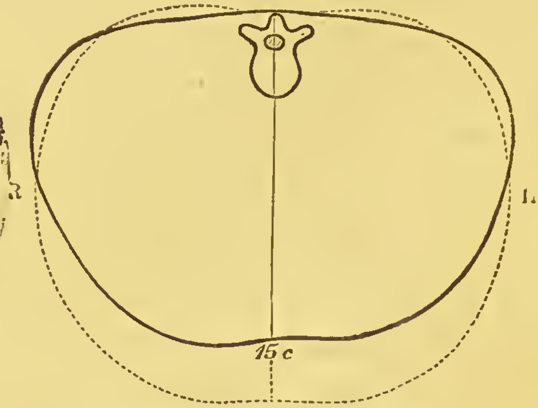
¶ II.—SUBTYPICAL SHAPES.

There are certain deviations from the typical shape of the chest which are present in a large number of persons free from any disease of the thoracic organs. And, partly for this very reason, but chiefly because these deformities do indicate pulmonary disease in the past, or a tendency to it in the future, they are worthy of all our attention. They are of five kinds, to wit, the alar or pterygoid, the flat, the transversely constricted, the pigeon, and the rickety chests.

i. *The Alar Chest.*—It has been known from of old that many persons predisposed to phthisis manifest their predisposition by an unnaturally small chest. Projection of the angles of the scapulæ, so as to look like wings, is one sign of the small capacity of these chests, which are

therefore called alar or pterygoid by Galen and Aretæus.[1] The thorax of phthinodes (persons predisposed to phthisis) is, as Galen says, nar-

Fig. 3.



Circumference = 59·5 centimeters.

Alar Thorax.

Tracing taken from a child. The dotted line indicates the shape of the chest of a healthy child of the same size. The circumferential measurement refers to the alar thorax.

row and shallow; the anteroposterior diameter is especially small. This diminution in caliber is brought about by drooping, or undue obli-

[1] "Phthinodes * planissime existunt qui thorace usque adeo sunt angusto atque non profundo, ut scapulæ alarum instar retro promineant, unde etiam tales pterygodes, hoc est alatos, vocitant." Galen. Comment. in Hippocr. Epid. i. pag. 62. edit. Kühn. 1828. Aretæus: Caus. et sign. morb. chron. i. 8. See also Boerhaave, Aphor. 1198, and Van Swieten's commentary.

quity, of the ribs, hence the shoulders fall, and the length of the thorax from above downwards is increased : it is the falling of the shoulders which causes the alar appearance. The pterygoid chest is often accompanied, as Aretæus says, with a prominent throat, due to a long neck, and the head being carried unduly forwards.

ii. *The Flat Chest.*—The diminution in capacity presented by the alar chest does not necessitate any great change in the outline which a horizontal plane of the thorax naturally presents. But sometimes not only the size of the sectional area but its shape also is changed, by the cartilages of the true ribs losing their curve and becoming straight. In which case the chest looks quite flat in front instead of being rounded, the horizontal ellipse is flattened from before backwards, nay sometimes the sternum is depressed below the level of the cartilages so that a section would be somewhat kidney-shaped, the cartilages being curved in the wrong direction. In other respects the flat chest mostly presents the characters of the alar thorax : but this is not always the case, inasmuch as the alar appearance is due to increased obliquity of the ribs and falling of the shoulders, conditions

which are not always present even in a well-marked flat chest, the diminution of capacity being otherwise brought about. Flat chests also indicate a phthisical disposition.

Both the phthinoid chests (alar and flat) are often modified in shape by the presence of the transverse constriction to be hereafter described. And both are, as Van Swieten says, essentially the same as the actually phthisical chest, but deformed to a less degree: moreover the loss of fat and muscle which occurs in phthisis makes all the characters described more obvious.

The phthinoid chests are Natural deformities, the tendency to which is born with the individual and inseparable from him. I now come to the Accidental deformities of the chest, those which have been produced by actual disease subsequent to birth, and not as a matter of necessity.[1]

iii. *Transverse Constriction of the Chest.*—A deformity from which few persons are wholly free. It consists in a depression, more or less deep, of the chest walls anteriorly, which passes

[1] "Naturalem formationem eam appello quæ sit cum pectore constricto, longo collo, et humeris alatis; accidentalem vero quæ sit cum curvitate seu distortione pectoris." Morton: Phthisiologia, 1689, ii. 1°.

outwards and slightly downwards, on both sides, level with the xiphoid cartilage, and ceases gradually towards the mid-axillary line.

Produced during childhood, the groove simply persists in after years. Its immediate cause is an impediment of some duration to the inspiration of air sufficient to distend the whole of the chest; the air fails to expand the bases of the lungs. Catarrh is that impediment in the vast majority of cases. Nor need the catarrh be at all severe, or the impediment at all great, inasmuch as a necessary concurrent cause of the groove is found in the yielding character of the ribs during infancy and early childhood, and especially of ribs rendered (as they so often are) præternaturally yielding by rickets.

When the impediment is severe and protracted the depression, although proportionally great, ceases to exist alone; other deformities are produced, and all together go to make up the pigeon breast.

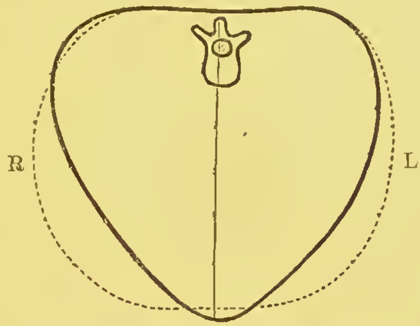
But when the deformity stops short of a pigeon breast, that is, when the depression is not so great as to involve the whole of the front base of the thorax from the xiphoid level downwards, it is the abdominal viscera which determine the position of the sulcus by maintaining

the expansion of the base of the chest. The depression occurs as low down as it can, namely, immediately above the upper surface of the abdominal viscera, or, what comes to the same thing, the sulcus corresponds to the vault of the diaphragm. This fact led Harrison to propose the sulcus as an easy means of determining the upper margin of the liver. But be it remembered that the groove indicates what was the upper margin of the liver in early life; and although no doubt the relationship of the parts concerned is scarcely altered in adult age, excepting from disease, yet it is clear that whatever changes the position of the liver after the furrow has been formed does not change the position of the furrow itself. However, for this very reason, albeit the groove and upper margin of the liver have ceased to correspond, the groove remains a valuable physical sign, indicative of what was the position and what the extent of the liver before the parts ceased to correspond.

iv. *The Pigeon Breast.*—The essential character of the pigeon breast is a straightening of the true ribs in front of their angles. Necessary consequences of this deviation from the natural shape are, first, that the sternum is thrown forwards, and next, that the greatest

transverse diameter of the chest recedes towards the costal angles, that is, that the hori-

Fig. 4.



Circumference = 57·5 centimeters.

PIGEON BREAST.

Tracing taken from a child of seven years. Dotted line indicates natural shape at same age.

zontal section tends to pass from the ellipse into the triangle.

The cause of the straightness of the ribs is a long existing or frequently recurring impediment to free inspiration while the ribs are plastic, that is, during childhood; and especially when they are unnaturally yielding, that is, when rickety. Chronic pulmonary catarrh (including whooping cough) and chronic enlargement of the tonsils are the common causes of pigeon breast: the deformity may be easily seen in process of formation by watching the

chest of a child during the long-drawn inspiration of whooping cough.

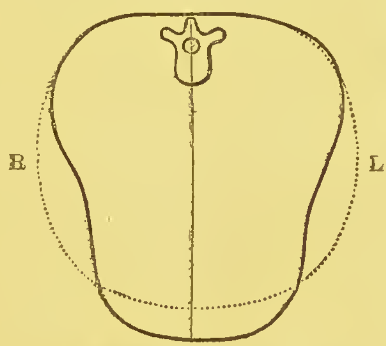
But obstruction to inspiration was pronounced to be the cause of the transversely constricted chest. And so a pigeon breast is mostly accompanied by a well-marked transverse sulcus: as a part of which transverse constriction, the xiphoid cartilage becomes bent back so as to form a more or less sharp angle at the bottom of the sternum; a condition which increases the similarity to a pigeon's breast. The transverse furrow, I say, is well-marked; in fact, very often there is a depression of the whole anterior half of the thorax below the xiphoid level; in which case, the straightening of the false ribs, in front of the angles, is carried to an extreme degree. This flattening below Harrison's sulcus is partly due to the fact that a deep inspiration involves the upper thorax chiefly.

v. *The Rickety Chest.*—Rickets, on account of the part which it takes in the generation of the transversely constricted and of the pigeon chest, has already been alluded to more than once: it remains to show how rickets alone may produce deformity of the chest.

Rickets is a disease of infancy, and infants cannot but have a respiration chiefly abdomi-

nal.[1] When the diaphragm descends, and rarefies the air contained in the lungs, the rickety ribs, not being able to hold out until the chest is completely distended by fresh air passing in through the glottis, yield in their softest places to the atmospheric pressure from without, and are bent inwards. Inasmuch as the softest parts of the ribs are at and near their costochondral articulations, a shallow longitudinal groove is

Fig. 5.



Circumference = 42·75 centimeters.

RICKETY CHEST.

Dotted line indicates shape of chest in an infant of about the same age.

formed on each side of the chest, parallel and a little external to the sternum : a groove which may be formed without the least direct impedi-

[1] Because of the circular shape of their thorax, which does not admit of further lateral expansion.

ment to the entry of air through the air-passages.

But in rickets the whole ribs are softened more or less, and hence when the causes of a transverse groove or of a pigeon breast are present in a rickety child, the resulting deformity is exaggerated.

Deformities of the chest which are purely rickety tend to disappear to a remarkable degree as the health improves: deformities of more complex origin are more permanent.

Article II.—Shape in Disease.

Having described the deviations from the natural shape of the chest which are compatible with a healthy state of its contents, I now come to those changes in shape which indicate disease of the thoracic viscera. Changes of this latter kind may be reduced into three classes, namely, bilateral, unilateral, and local. The former two classes of change indicate disease of the lungs or pleuræ : the last class of change may be caused by disease of lungs, heart, serous membranes, or mediastina.

¶ I.—BILATERAL CHANGES IN SHAPE :

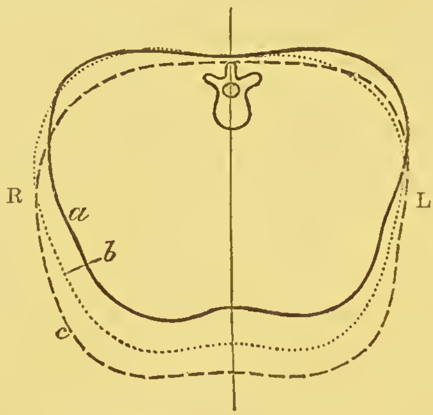
are of two kinds, enlargement and diminution.

i. *Bilateral Enlargement.*—By the deepest inspiration (in other words, by the greatest elevation and rotation of the ribs) no considerable modification can be produced in the proportion between the length of the two axes of the horizontal ellipse. In order to render further enlargement of the thorax possible the ribs must change their shape ; they become more curved : the axes of the ellipse tend to become equal ; the ellipse tends to pass into the circle ; changes which are explained by the fact that of all figures possessing a periphery of fixed and certain length, the circular is that which includes the greatest area ; depart from the circle in any way and the area becomes less. It is a fact which must be clearly understood ; that the chest admits of an enlargement far beyond that which can be produced, when the lungs are healthy, by the deepest inspiration. It is quite a mistake to suppose that the bilaterally enlarged chest is merely in a state of permanent inspiratory expansion. The experiment indicated by the diagram on the next page is decisive upon this point. Moreover Freund[1] has shown

[1] Der Einfluss der primären Erkrankungen des knorpligen Thorax auf Entstehung gewisser Lungenkrankheiten. Würzb. Verhandl. 1859. Bd. ix. p. 223. See also Rud. A.

that, in an emphysematous chest, there is sometimes an actual increase in the length and breadth of the costal cartilages.

Fig. 6.



Horizontal section of chest of a child two years old.

a = chest at rest.
b = chest after fullest expansion possible of lungs.
c = chest after forcible injection of air into both pleural cavities.

Anteroposterior diameters :

a = 10·2c. *b* = 11·7c. *c* = 13·2c.

Circumferences :

a = 47·5c. *b* = 48c. *c* = 48·5c.

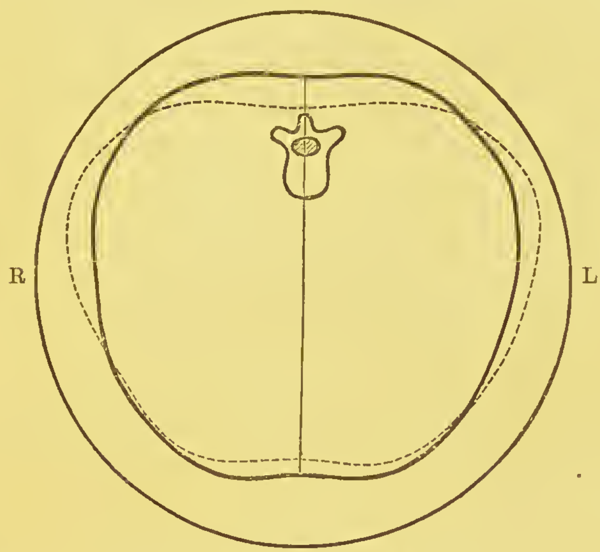
Enlargement of the chest signifies enlargement of its contents : the only possible causes of bilateral enlargement are emphysema of the lungs (whether hypertrophous and permanent,

Vogel : Observationes binæ de Asthmate singulari ex cartilaginum costarum ossescentia. Gottingæ, 1773.

or mere temporary distension of the air-vesicles,) and a large effusion of fluid into both pleuræ. Now the latter state is incompatible with life, and so a bilaterally enlarged chest, and the chest of emphysema come to be convertible terms.

In a well-marked case of emphysema, then,

Fig. 7.



Bilateral Enlargement of Emphysema.

Inner line = emphysematous chest.
Outer line = a circle drawn to show how nearly the emphysematous approaches the circular shape.
Dotted line = natural adult chest.
Actual measurements in centimeters :

Circumference	= nat.	89·	emphys.	87·75
Transverse	= ,,	29·6	,,	27·25
Anteroposterior	= ,,	22·25	,,	25·4

the thorax is in a state of permanent distension beyond what could have been produced

during health by the deepest possible inspiration. The chest is, as Laennec says,[1] almost cylindrical or globular, arched before and behind. The arching is usually most marked in the sternum, and is simply the result of the fact that the sternum is less able to move forwards above than below: the manubrium and body of the breast-bone become bent at an angle, the angulus Ludovici. But sometimes the spine is much more arched than the sternum, and this may be the case to such an extent, in a thorax highly emphysematic, that the sternum shall be nearly straight, and the front of the chest apparently flat, in consequence of the shoulders being thrown forwards by the stooping of the vertebræ.

The bilateral enlargement sometimes involves the whole length of the thorax, and then the cartilages of the false ribs are everted, and the costal angle is greatly increased in size. But sometimes the enlargement affects the chest above the xiphoid level only; the parts below being tolerably natural, or even depressed: when they are depressed, the transverse constriction is well marked, and the costal angle

[1] Auscultation Médiate, 2me edit. 1826, i. 307.

is diminished in size. The causes of the depression are the same as described when speaking of pigeon breast; very little air enters, what does enter goes to the upper parts; the respiration is superior-thoracic.

Kyphosis (angular curvature) of the dorsal spine, whether senile, or due to the carrying of heavy weights upon the shoulders, or to caries of the vertebræ, is attended by a shape of chest which strongly simulates emphysematous enlargement.

It is instructive to compare the raising of the shoulders and the non-prominence of the shoulder-blades with the opposite conditions in the opposite form of chest, the pterygoid.

ii. *Bilateral Diminution.*—The diminution is greater than can be produced in a healthy chest by the deepest expiration. The characters of the chest are in all points the same as those of the flat phthinoid chest but carried to a greater pitch. Phthisis is the only disease in which great diminution of both sides of the thorax occurs; as Aretæus says excellently well,[1] the chest becomes broader (*i. e.*, in the transverse diameter), yet the patients stand in need of its

[1] Caus. et sign. Mb. diut. i. 10.

being broader still (*i. e.*, in the anteroposterior diameter).

¶ II.—UNILATERAL CHANGES IN SHAPE.

These, like the bilateral, consist in enlargement and diminution.

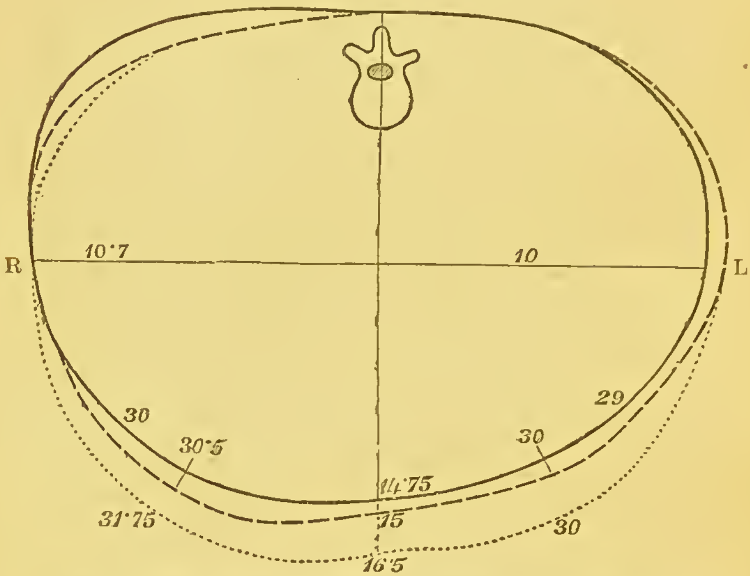
i. *Unilateral Enlargement.*—The notes of bilateral enlargement already given are applicable to enlargement of one side only of the chest. The side enlarged, compared with the other, will present these characters : shape rounder ; transverse semidiameter shorter, and anteroposterior diameter longer ; length, from above downwards, diminished ; shoulder raised ; spine curved towards unaffected side. I say that the length of the chest from above downwards (the vertical diameter) is diminished, and this is true, provided that the floating ribs be excluded from consideration : let any one who doubts this statement inject the pleura of a dead subject with air, and watch the changes which ensue.[1]

[1] Gueneau de Mussy (L'Union médic. 1866, No. 95) maintains that the lower ribs are depressed by a pleural effusion : I have not yet been able to convince myself that this is so, or is so to such an extent, at least, as to be a valuable physical sign. I do not deny that in subdiaphragmatic tumours the lower ribs may be raised.

The anteroposterior enlargement becomes very obvious when the physician stands behind the patient so as to look obliquely over his shoulders and the front of his chest. In children the best notion of the enlargement and roundness of the affected side is gained by grasping both sides with the two hands, the thumbs being placed tip to tip upon the spines of the vertebræ. Circumferential measurements of the two sides are often made, but be it remembered, first, that considerable increase in the sectional area of the chest may occur, and the length of the periphery remain the same, by the passage of the elliptical form into the circular : and next, that the displacement of the mediastinum, which accompanies unilateral enlargement, thrusts the heart into the unaffected side. Add this consideration too, that the walls of the healthy side must follow the anteroposterior projection of the diseased side : and then it will be plain why, as a matter of fact, the perimeter of the expanded side often measures very little more, nay even less, than that of the side which is not diseased. The cyrtometer alone, by indicating shape as well as circumference, affords us the true means of recording the amount of a unilateral enlargement.

The causes of bilateral and unilateral enlargements are the same, namely, increase in the size

Fig. 8.



Unilateral enlargement of chest (right side); artificially produced by injecting air into the right pleural cavity.

Unbroken line = outline before injection.
Broken line = outline after moderate distension.
Dotted line = outline after extreme distension.

Figures, at bottom of vertical line indicate the anteroposterior diameters: along horizontal line indicate transverse semidiameters: remaining figures indicate right and left semicircumferences.

Compare fig. 18, p. 209.

of the lung, and effusion of fluid into the pleura. Increase in the size of one lung occurs in vicarious hypertrophy compensatory of chronic disease whereby the other lung is put out of

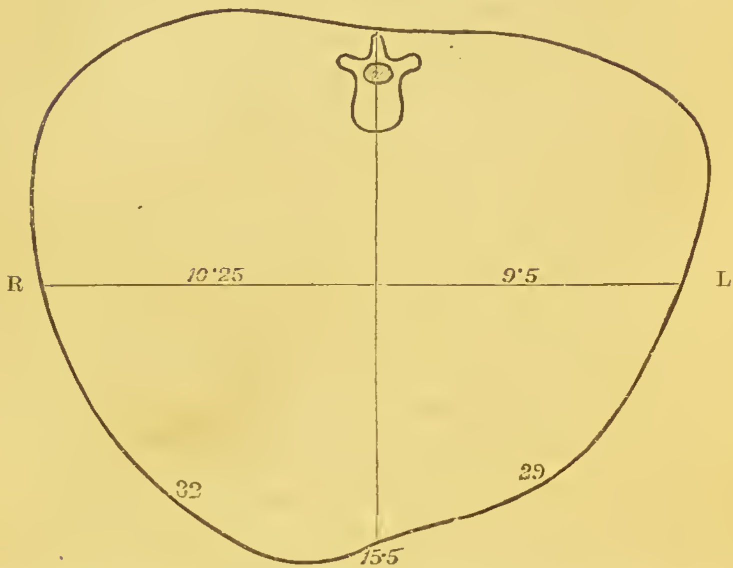
play; in rapidly growing soft cancer of the lung; and, according to Woillez, in active sanguineous fluxions upon the lung: unilateral hypertrophous emphysema, the other lung being healthy, is impossible. Effusion of fluid into the pleura, however, causes the greatest enlargement: inflammatory effusion, pneumothorax, extensive hæmothorax.

ii. *Unilateral Diminution.* — In unilateral shrinking of the chest the circumference and the anteroposterior diameter are diminished; the transverse diameter increased; the side looks flat before and behind, having lost its rounded shape and become angular; the ribs are closer together than natural; the shoulder lower; and the spine is curved towards the healthy side. The perimeter of the contracted side is diminished; and, when there is vicarious enlargement of the other lung, the difference between the circumference of the two sides becomes very great.

Unilateral diminution of the chest, when chronic, is always attended by an adherent pleura; a condition itself the result of past pleurisy, of phthisis, or of cancer. An acute unilateral shrinking of the chest occasionally occurs in children, as a consequence of collapse

of one lung due to an obstruction of the main bronchus.

Fig. 9.



Unilateral retraction of chest: consequent upon cirrhosis of left lung in a girl of fourteen. Figures as in last sketch.

Scoliosis sometimes induces a shape of chest which, when regarded from the front or from the back alone, strongly simulates unilateral contraction.

¶ III.—LOCAL CHANGES IN SHAPE,

namely local bulging and shrinking.

i. *Local Bulging.*—Bulging of a part of the

chest walls is met with in circumscribed pleural effusions, in pericardial effusions, in large phthisical excavations, in hernia of the lung, in hypertrophy and dilatation of the heart, in pointing empyemata, in aneurysms, in cancerous, hydatid, and other tumours.

The bulging indicative of cardiac or pericardiac enlargements demands further notice. It occurs between the third and seventh cartilages on the left side, and extends from the left nipple line to the sternum or even to the right nipple line. Paralysis of the intercostal muscles probably ensues, as Gendrin thought, when the bulging becomes great.

ii. *Local shrinking.*—Shrinking of a part of the chest walls, when due to intrathoracic disease, is usually attended, like unilateral contraction of the whole chest, by pleural adhesions. Shrinking of the apex of one side is very common in phthisis. There is a condition, frequently met with, which has never been explained, and which is difficult of explanation; I refer to a great cup-like depression of the lower part of the sternum and of the attached cartilages. I have known this deformity to follow unilateral pleurisy, and yet to be perfectly symmetrical. Croup, in infants, is often attended

by enormous inspiratory recession of the whole corpus sterni below the angulus Ludovici: whence I infer that a chronic catarrhal obstruction to inspiration may cause, in some cases, this crateriform depression instead of the more usual pigeon breast: the angle produced by the horizontal constriction (page 20) occurs higher up than usual. Rickets, no doubt, would be auxiliary. A slight degree of a similar deformity is found in many shoemakers.

SECTION II.

MOVEMENTS OF THE CHEST.

These are of three kinds, movements of respiration, movements of the heart, and movements wholly præternatural.

Article I.—Movements of Respiration.

¶ I.—IN HEALTH.

Healthy breathing, and its varieties according to age and sex, are subjects so fully discussed by physiology that they may be passed by in this place. The construction of an instrument, which would apply the principle of the sphygmograph to the registration of the respiratory movements,

has engaged the thought of Marey,[1] Gerhardt,[2] Hawksley,[3] and Sanderson.[4] The difficulty of breathing naturally, while undergoing an examination of the chest, must frequently prove an insuperable bar to the use of a stethograph. The spirometer [5] cannot be said ever to have taken rank among the means of physical diagnosis. Whether the anapnograph [6] will be more useful remains to be seen.

¶ II.—IN DISEASE.

Mere increase and mere diminution of respiratory movement, bilateral, unilateral, or local, scarcely demand a long notice. Perversions in the character of the movement are more important: they may be arranged under five heads, inspiratory dyspnœa, expiratory dyspnœa, non-

[1] Mouvements dans les Fonctions de la Vie, 1868, page 163.

[2] Lehrbuch der Auscultation und Percussion, 1866, page 103.

[3] Proceedings Royal Med. Chir. Soc. vol. vi. page 203, 1869. Also, Lancet, June 12, 1869.

[4] See Med. Times and Gaz., Sept. 4, 1869, page 291.

[5] John Hutchinson: On the Capacity of the Lungs and on the Respiratory Functions, with a view of establishing a precise and easy method of detecting disease by the Spirometer. Med. Chir. Trans. vol. xxix. 1846.

[6] Bergeon et Kastus: Gazette hebdom. 1868; 37, 39, 40.

expansive inspiration, respiration wholly thoracic, and respiration wholly abdominal.

i. *Inspiratory Dyspnœa.*—It is when the chest-walls are yielding that inspection affords the most convincing sign of inspiratory dyspnœa; namely, a recession, more or less deep, of the front of the thorax below the level of the nipples. The causes and mechanism of this recession are identical with the causes and mechanism of the transversely constricted chest (page 17). But I speak now of an amount of constriction so great as to be incompatible, for any length of time, with life. Inspiratory dyspnœa, such as this, occurs in obstruction to the passage of air through the larynx or trachæa, in per-acute œdema of the lungs and hydrothorax, and in infants or rickety children even during a common pulmonary catarrh. In the last case temporary bilateral enlargement of the chest, above the transverse sulcus, mostly concurs with the depression of the parts below. In obstruction to the large air-passages the supraclavicular regions usually partake in the recession.

ii. *Expiratory Dyspnœa.*—In this condition the expiratory movement is exceedingly laborious and prolonged. Expiratory dyspnœa is present along with any great impediment to

expiration in the upper air-passages; and also occurs whenever the expiratory power of the lungs is diminished, as in emphysema and congestion.

iii. *Non-expansive Inspiration.*—The chest-walls are elevated powerfully, yet expanded little or not at all. This sign may occur, on one side or on both, whenever the texture of the lung is impermeable to air, or non-expansible (as in pleurisy with effusion, pneumothorax, dense pleural adhesions, phthisis, and cancer), or when the thorax cannot be dilated more than it is (as in emphysema). The application of the hand to the chest is very serviccable in the detection of non-expansive inspiration.

iv. *Respiration wholly Thoracic.*—The female type of respiration carried to the extreme. Occurs in diseases which interfere with the action of the diaphragm: paralysis of the diaphragm, great pericardial effusion, peritonitis, ascites, abdominal tumours.

v. *Respiration wholly Abdominal.*—The male or infantine type of respiration carried to the extreme: respiration being performed almost wholly by the diaphragm. Occurs bilaterally in spinal paralysis, and in tetanus; unilaterally in pleurodynia, pleurisy, phthisis.

Article II.—Movements of the Heart.

The chief movement of the heart, detected by inspection, is the Impulse. Systolic recession of certain interspaces is sometimes visible.

¶ I.—MOVEMENTS IN HEALTH.

i. *The Impulse.*—The chief cause of the impulse was, I believe, first suggested by Dr. Billing (page 140); namely, increase in the antero-posterior diameter of the heart during the ventricular systole. The part of the heart which strikes the chest wall is a spot upon the anterior surface of the right ventricle, about three-quarters of an inch above the apex: the apex itself being separated from the thoracic parietes by a layer of lung which is a finger broad. After what has been said concerning the cause of the impulse I need scarcely remark that it is always exactly systolic.

The Position of the impulse corresponds with the fifth left interspace, midway between the nipple and the parasternal lines. In children, under eleven years of age, the heart frequently lies high, so that the impulse occurs in the fourth interspace, and rather more external than in

adults, that is to say, in the nipple line. In many old people the heart lies low, so as to beat against the sixth interspace. By a deep inspiration the impulse can be depressed half an inch. By lying upon the left side the position of the impulse shifts to the nipple line, or beyond it.

The Extent of the visible impulse is small, not greater than a square inch.

The Force of the impulse is best estimated by palpation (page 47).

ii. *Recession of the chest walls.*—Is occasionally perceptible, during the systole, in the third, fourth, or even the fifth intercostal spaces, close to the sternum: this, especially in persons who are very thin.

¶ II.—MOVEMENTS IN DISEASE.

i. *The Impulse.*—The Position of the impulse may be changed by whatever enlarges the heart or changes its position.

In enlargement of the left ventricle, the impulse occurs to the left of the nipple line, in the sixth or even the seventh interspace. In enlargement of the right ventricle, the impulse is rather increased in extent, to the right of the parasternal line, than altered in position.

Displacement of the heart to the left may be so great as to cause the impulse to beat in the axillary line, and in any interspace from the second to the fifth or sixth. The causes of this kind of displacement are the following: diseases of the abdomen (ascites, tympanites, tumours) whereby the diaphragm is raised, and the heart comes to lie more horizontally than is natural; effusions of air or liquid into the right pleura; tumours to the right of the heart; retraction of the left lung, whether by phthisis, cirrhosis, or old pleurisy.

Displacement of the heart to the right may cause the impulse to be felt anywhere to the left of the right nipple line, in any interspace from the fourth to the seventh, or in the epigastrium. The causes are the same as those of displacement to the left, excepting abdominal enlargement.

Let it not be supposed, however, that, when the heart is enlarged or displaced, the impulse always continues to correspond with the apex of the heart. Dr. Powell[1] has shown that, when displacement to the right occurs, although the chief impulse may be felt under the right nipple,

[1] On displacements of the heart. Brit. Med. Journal, July 17, 1869.

yet this is not because the apex beats there. On the contrary the heart can never be displaced, towards the right, into more than a vertical position; the long axis of the ventricles never comes to slant downwards to the right. When the impulse is felt to the right of the sternum, some part of the right ventricle other than the apex strikes against the chest; and this part is usually the conus arteriosus.

The Extent of the impulse (meaning thereby actual contact of the heart with the chest wall) may be so increased as to be visible in the second, third, and fourth intercostal spaces; and then the movement sometimes takes on a fluctuating or peristaltic character. The causes of a too extensive impulse are these: enlargement of the heart, the lung being pushed aside; shrinking of the lung; and whatever presses the heart against the chest-wall, for instance, a narrow chest, or a tumour in the posterior mediastinum, especially an aneurysm of the descending aorta.[1]

ii. *Recession of the Chest-wall.*—In hypertrophy of the heart, the recession of the chest above the apex-beat is much more frequently met with than in health. The apex-beat itself is some-

[1] The subject of Epigastric Pulsation is discussed in the Appendix, page 171.

times strongly dimpled inwards during the systole; a sign which usually indicates the presence of pericardial adhesion, combined with pleural adhesions over the pericardium; for, the heart, being thus fixed to the diaphragm and the mediastinum, manifests the shortening which occurs during the ventricular systole, by dragging the chest walls inward (page 252).

Article III.—Movements wholly Præternatural.

Movements, that is to say, which have no counterpart in the healthy state. The pulsation of aneurysmal tumours is of this kind.

CHAPTER III.

PALPATION.

SECTION I.—LUNGS AND PLEURÆ:

Art. I. Vocal Thrill:

¶ I. In Health.

¶ II. In Disease:

i. Diminished.

ii. Increased.

Art. II. Friction Fremitus.

Art. III. Fluctuation.

i. Produced by observer.

ii. Produced by heart.

SECTION II.—HEART AND PERICARDIUM:

Art. I. Impulse:

¶ I. Systolic:

i. Force diminished.

ii. Force increased.

¶ II. Diastolic:

i. Back-stroke of heart.

ii. Pulmonary diastolic.

¶ III. Præsystolic.

Art. II. Valvular Thrills:

i. Aortic.

ii. Pulmonary.

iii. Mitral.

PALPATION, or the application of the hand to the surface of the chest, is often useful as a means of confirming the notions acquired by the eye concerning the shape, size, or amount of movement of any part of the thorax. But palpation has an independent value, and can detect thrills and impulses which are imperceptible to the eye. These signs will be considered in order as they relate to the lungs and pleuræ, the heart and pericardium, and the large bloodvessels.

SECTION I.

LUNGS AND PLEURÆ.

Article I.—The Vocal Thrill.

¶ I.—IN HEALTH.

When a person speaks, a distinct tremulous sensation may often be felt upon the surface of the chest: the vocal fremitus or thrill. The intensity of the thrill is in direct proportion to the depth and loudness of the voice, and there-

fore is best marked in adult men, and is usually absent in women and children. The reason of this is clear: only in low-pitched notes are the vibrations sufficiently far apart to be perceptible to the hand. The thrill is strongest over the lungs, and is rapidly weakened as we pass away from them: the thoracic walls conduct the thrill well when they are not very fat or œdematous. Vocal fremitus is naturally more intense on the right side than on the left.

In examining the vocal thrill it is best to make all patients repeat the same sound: that which is most suitable for this purpose is the pure laryngeal sound ah.

¶ II.—IN DISEASE.

i. The vocal thrill is diminished or abolished by whatever separates the lung from the chest-wall, and by whatever renders the lung quite impermeable to air. An effusion into the pleura, whether of liquid or gas, is the commonest cause of loss of vocal fremitus; but, unless the effusion be great, the thrill is not wholly abolished, being conducted by the thoracic walls. Solidification of the lung does not materially diminish vocal thrill except the solidification be very dense. Wherefore ordinary pneumonic and phthisical

consolidations do not lessen the thrill, and may even increase it: but pneumonic exudation which packs the lung hard, massive phthisical solidification, and cancer, these abolish vocal fremitus, unless indeed a large bronchus be in intimate connection with the solid mass.

ii. The vocal thrill is increased by consolidation which is not too dense, and which is traversed by large open air-tubes.

The thrills produced by cough and rales are of no practical value.

Article II.—Friction Fremitus:

May occasionally be felt, but is of no practical importance.

Article III.—Fluctuation.

Fluctuation of a liquid effusion in the pleura may sometimes occur.

i. Fluctuation produced by striking the chest is a physical sign wholly superabundant, inasmuch as a complete diagnosis will have been attained long before the disease has gone so far as to yield the sign of fluctuation. When the intercostal spaces are widened and rendered tense, fluctuation may sometimes be produced

and felt by two fingers placed far apart in the same interspace. The splash of hydropneumothorax may be often felt, but is much better heard.

ii. Fluctuation of a pleural liquid effusion is occasionally produced by the action of the heart: for the diagnosis of this condition see the chapter on empyema (page 216).

SECTION II.

HEART AND PERICARDIUM.

Article I.—The Impulse.

¶ I.—SYSTOLIC IMPULSE.

Palpation will serve to confirm the information yielded by the eye as to the position and the extent of the impulse of the heart against the chest wall. Sometimes the impulse is palpable when it is not visible.

Palpation moreover enables us to say whether the impulse is weaker or stronger than natural.

i. The force of the impulse is diminished by whatever weakens the heart, and by whatever separates it from the chest-wall. Great diminution in the force of an impulse which has been heaving is an important sign of asystolism (page 237).

ii. The force of the impulse when increased, that is to say, a heaving impulse, is much better appreciated by applying the stethoscope and the head to the chest, than by the hand alone. A knowledge of this fact was the immediate forerunner of the discovery of auscultation (page 99). When the heaving impulse is well marked, it is able to overcome the greatest possible amount of pressure, and may be regarded as a sure and certain sign of cardiac hypertrophy. The mere knocking of a palpitating heart is easily distinguished from true heaving.

The force and extent of the impulse are much influenced by the thickness of the thoracic parietes, the position of the body, the tone of the heart, and the relative situation of the lungs.

The Cardiograph is an instrument, invented by Chauveau and Marey,[1] whereby the movements of the cardiac impulse may be registered. A cup, containing air, and communicating by

[1] Chauveau et Marey: Démonstration nouvelle du mécanisme des mouvements du cœur par l'emploi des instruments enregistreurs à indications continues. Mém. de l'Acad. de Méd. Paris, 1861. Marey has published several later works upon the same subject. See also an article by Lorain: Cardiographie. Nouv. Dict. de Méd. vol. vi. 1867.

means of a caoutchouc tube with a small chamber which acts upon a graphic mechanism similar to that of the sphygmograph, is placed tightly upon the chest so as to surround the impulse: the movements of the impulse are conveyed, by the air contained in the apparatus, to the registering lever. Hitherto more service had been done to physiology than to medicine by the cardiograph.[1] It has confirmed, in the most positive manner, the truth of the following propositions:—

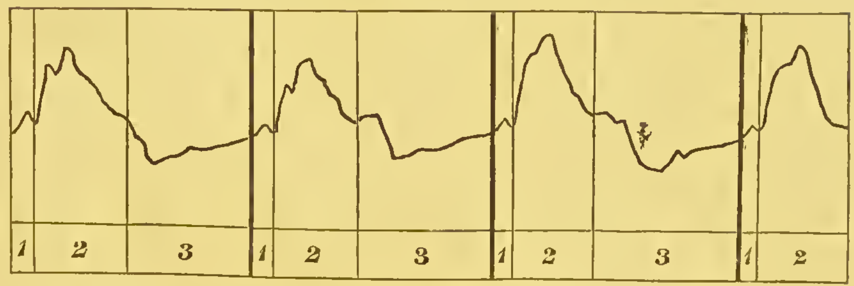
That the contraction of the auricles immediately precedes that of the ventricles: that the systole of both ventricles is exactly synchronous: that the impulse and the ventricular systole exactly coincide, both in time of occurrence and in duration: that the second sound follows immediately upon the ventricular systole: that the auriculo-ventricular valves vibrate during the greater part of the ventricular systole.

Copies of cardiographic tracings are annexed, inasmuch as they afford the best diagram of the sequences of cardiac action.

[1] See chapter on Aortic Regurgitation.

Fig. 10.

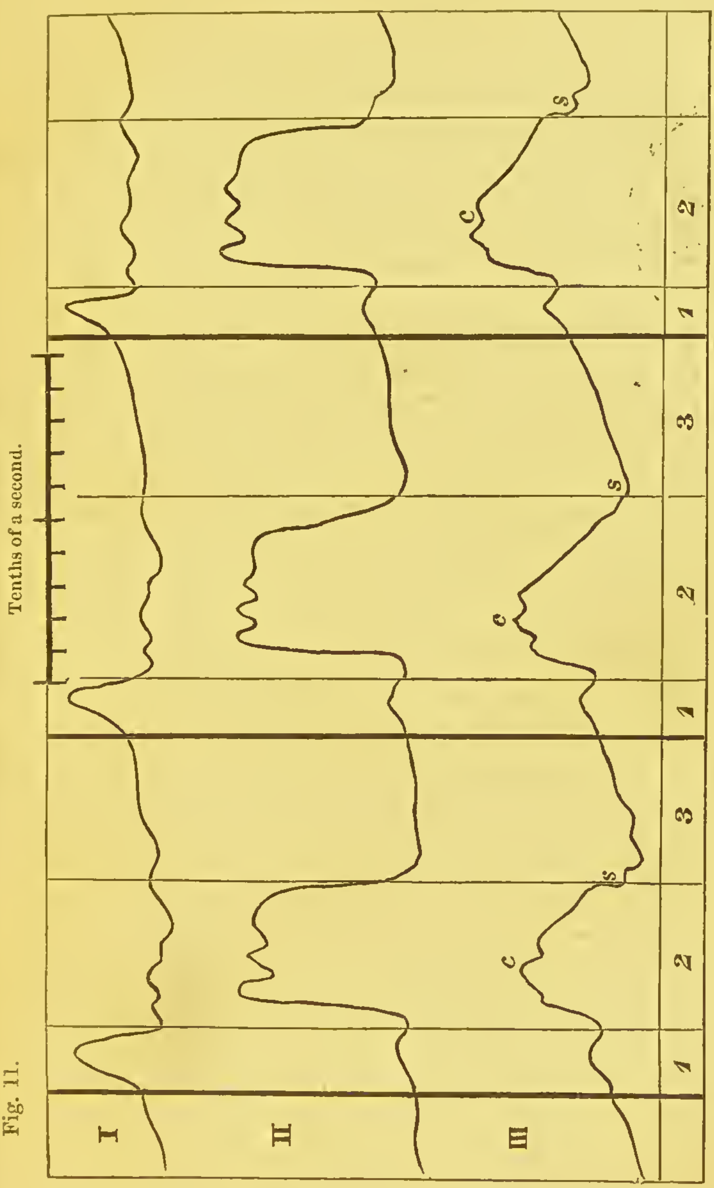
TRACING AFFORDED BY IMPULSE OF HUMAN HEART.



1 = præsystolic period........ ...contraction of auricles.
2 = systolic period ....contraction of ventricles.
3 = diastolic period ....heart at rest.

Fig. 11.

TRACINGS AFFORDED BY HORSE'S HEART.



I. Tracing afforded by auricle.
II. Tracing afforded by ventricle.
III. Tracing afforded by impulse.
c = vibration of cuspid valves.
s = vibration of sigmoid valves.

¶ II.—DIASTOLIC IMPULSE.

Diastolic cardiac impulses are of two kinds:

i. Sudden relaxation of the ventricles, after a powerful contraction, sometimes causes a sort of diastolic impulse; the back-stroke, as it was called by Hope. This most frequently occurs in hypertrophied hearts.

ii. Sudden closure of the pulmonary sigmoid valves sometimes causes a sharp invisible diastolic impulse, which is to be felt at the second left interspace close to the sternum. This sign indicates the presence of solid lung over the pulmonary artery, or that the lung has shrunk away from it, or that there is an unusually high pressure in the artery. The last cause is the commonest; is usually present in disease of the mitral orifice, and occasionally even in perfect health; and is attended by a loud pulmonary second sound.

¶ III.—PRÆSYSTOLIC IMPULSE.

The contraction of the auricles, when they are much hypertrophied, may give rise to a short præsystolic impulse at the base of the heart.

Article II.—Valvular Thrills.

A valvular thrill is a quivering sensation felt by the hand applied to the region of the heart in certain forms of disease. The likeness to the purring of a cat led Laennec[1] to invent the name "frémissement cataire" for the phænomenon: by its discoverer, Corvisart, it had been previously described under the term "bruissement."[2] For a thrill to be of value as a physical sign it must be well marked: what may be considered a well-marked thrill can be learnt by experience alone. Thrills are of the following kinds:—

i. Thrills felt in the second right intercostal space, close to the sternum, or at the episternal notch: they are systolic, or, in rare cases, diastolic.

ii. Thrills felt in the second left intercostal space, or over the third left costal cartilage, close to the sternum: systolic.

iii. Thrills felt at the apex-beat of the heart: not mere tremulousness of the impulse, but well-marked thrills: they are mostly præsystolic, going immediately before the impulse, and

[1] Ausc. Méd. 2me éd. ii. 448.

[2] Maladies du Cœur, 2me éd. 1811, p. 232.

brought to a close thereby: occasionally they are systolic.

iv. Thrills felt over the fourth left costal cartilage, or in the fourth left interspace, close to the sternum: præsystolic.[1]

Systolic and præsystolic thrills are indicative of obstruction to the passage of the blood through a valvular orifice. Thrills, at the base of the heart and to the right of the sternum, indicate obstruction at the aortic orifice, and are usually systolic, because the blood is passing through the orifice at that time. When diastolic, a regurgitation of blood through incompetent valves causes the thrill. In like manner, systolic thrills to the left of the base of the heart indicate pulmonary obstruction. An apex thrill is due, as Corvisart knew perfectly well, to obstruction at the mitral orifice; and is præsystolic because the blood passes through that orifice immediately before the ventricular systole, that is to say, during the auricular systole. In like manner, præsystolic thrills felt over the right ventricle indicate tricuspid obstruction: but they are rare.

[1] Gairdner, Edinb. Med. Journal, v. 871. Haldane, Edinb. Med. Journal, x. 271.

Article III.—Friction Fremitus.

It is not at all uncommon to be able to feel the rubbing of pericardial exudations: a matter of small practical value. Friction fremitus is usually very unlike valvular thrill.

Article IV.—Fluctuation.

In chronic copious pericardial effusions fluctuation may sometimes be felt, or even seen: an observation as old as Senac.[1]

SECTION III.

LARGE VESSELS.

The impulses and thrills produced in the large bloodvessels will be considered in the chapter on Thoracic Aneurysms.

[1] Corvisart, p. 49.

CHAPTER IV.

PERCUSSION.

SECTION I.—INTRODUCTORY.

Art. I. Historical.

Art. II. Method of Percussion:

i. Immediate percussion.

ii. Mediate percussion.

Art. III. Theory of Percussion:

¶ I. Percussion Sound:

Class I. Resonant Sounds:

I. Loudness.

II. Duration.

III. Pitch.

Physical Conditions of perfect Resonance.

IV. Clearness.

Physical Conditions of muffled Resonance.

Class II. Non-Resonant Sounds.

Physical Conditions of Non-resonance.

Appendix: Metallic Sounds:

I. Amphoric Resonance.

II. Cracked-pot Sound.

¶ II. Percussion Resistance.

Superficial and deep Percussion.

SECTION II.—PERCUSSION OF THE CHEST IN HEALTH.

Art. I. The Pulmonary Region:

¶ I. Pulmonary Resonance and Resistance:

i. Typical.

ii. Sub-typical: modifications compatible with health.

¶ II. Extent of Pulmonary Region.

Art. II. The Cardiac Region:

i. Superficial Area.

ii. Deep-seated Area.

Art. III. The Mediastinal Region.

SECTION III.—PERCUSSION OF THE CHEST IN DISEASE.

Art. I. The Pulmonary Region:

Auenbrugger's Dictum.

¶ I. Pulmonary Resonance:

Class I. Increased Resonances.

Class II. Diminished Resonanccs.

Appendix: Metallic Sounds.

¶ II. Pulmonary Resistance.

¶ III. Extent of Pulmonary Region.

Art. II. The Cardiac Region:

i. Area diminished.

ii. Area increased.

Art. III. The Mediastina.

SECTION I.

INTRODUCTORY.

ARTICLE I.—HISTORICAL.

PERCUSSION, or the art of striking a part of the body so as to beget a sound useful for the discovery of disease, has been prac-

tised from the earliest times. Employed at first in the diagnosis of abdominal diseases (to distinguish tympanites from ascites [1]), it was not until the middle of the last century that percussion was applied to the discrimination of the diseases of the chest. This important extension of the powers of percussion we owe to Auenbrugger, who in 1761 published a small book descriptive of his method.[2] When we hear of the reception which Auenbrugger's contemporaries in the city of Vienna gave to his discovery it is difficult to restrain a feeling of anger. The medical dictators of the day, Van Swieten and De Haen,[3] unjustly ignored per-

[1] This most ancient means of diagnosis is probably at least as early as Hippocrates. During the three busy centuries after Hippocrates, the Greeks invented the word tympanites, which was in familiar use by the time of Celsus.

[2] Leopoldi Auenbrugger, M.D., Inventum novum ex percussione thoracis humani ut signo abstrusos interni pectoris morbos detegendi. Vindobonæ, 1761. This edition is extremely rare: a reprint was published at Graz in 1867.

[3] De Haen himself has suffered in a similar manner. The admirable papers, which appeared from time to time in the Ratio Medendi, upon the use of the thermometer in medicine, were forgotten for eighty years; and the leading facts of clinical thermometry, perfectly well known to De Haen, have been rediscovered in our own day.

cussion altogether: less exalted personages stupidly confounded it with the Hippocratic method of succussion. Auenbrugger tells us that he was prepared to meet with envy, hatred, and calumny; we know that he did meet with what is harder to bear, simple neglect: however the scanty records of the man's life justify the belief that he passed from one social deed to another social deed, and found his happiness in doing so.[1] It was worthy of Stoll that he at least should not fail to acknowledge the value of percussion;[2] but it was Corvisart who, guided by his favourite author, Stoll, may be said to have discovered Auenbrugger. For many years Corvisart practised percussion at the Charité of Paris, and finally, in 1808, he published a translation of the Inventum Novum.[3] Henceforth the importance of percussion was undisputed.

The principles of the Inventum Novum are two: first, that the sounds produced by per-

[1] Leopold Auenbrugger, der Erfinder der Percussion des Brustkorbes, und sein Inventum Novum: von Prof. Dr. Clar. Graz, 1867.

[2] M. Stoll. Prælectiones in Morb. Chron. 1788. i. 86.

[3] Nouvelle Méthode pour reconnaître les maladies internes de la poitrine, par la percussion de cette cavité, par Avenbrugger, ouvrage traduit du latin et commenté par J. N. Corvisart. Paris, 1808.

cussion must be regarded simply as acoustic phænomena, and named accordingly; secondly, the sounds are to be explained by reference to corresponding physical states, that is to say, to the presence or absence of air in the part percussed.[1] Anenbrugger's doctrines remained unassailed until Piorry, in 1826,[2] sought to establish a new theory, founded upon the principle that every organ yields to percussion a peculiar sound, "chaque organe étudié par le plessimétre a un son spécial." Hence a series of typical sounds: osteal, femoral, jecoral, cardial, stomacal, coecal, intestinal, pulmonal, humoral, metallic (sound of water and air), and hydatid. This scheme, however, is now discarded by its author, who has proposed another in its stead: we will not forget that Piorry invented the pleximeter. In 1839, Skoda[3] published his theory, based upon two principles: "we must first determine every possible variety of percussion-sound, and ascertain the conditions on

[1] Inventum Novum, §§ 17, 18, scholia.

[2] Paper read before the Parisian Academy of Medicine in that year.

[3] Abhandlung über Percussion und Auscultation. Wien, 1839. This work reached a sixth edition in 1864. A translation of the fourth edition, by Dr. Markham, was published in 1853.

which each variety depends; and then endeavour to reconcile our observations with the well-ascertained laws of sound." Wherefore, in the first place, Skoda restores Auenbrugger's principle of nomenclature, and distinguishes percussion-sounds according as they are full or scanty, clear or dull, tympanitic or not tympanitic, high or low; epithets which will be explained hereafter. In the next place, Skoda developes Auenbrugger's second principle by maintaining that "the differences in the percussion-sounds of sundry organs [except bone and cartilage] depend, not upon any peculiarities in the organs themselves, but upon differences in the quantity, distribution, and density of the air present in the regions where these organs lie; and upon the force of the blow which percussion can exert upon this air." The doctrines of Auenbrugger and Skoda are in essence the doctrines of the present day.

Article II.—Method of Percussion.

i. *Immediate Percussion.*—This was the method employed by Auenbrugger, and may be described in his own words: "Let the chest be percussed by the tips of the fingers drawn to-

gether side by side and stretched out straight; let the chest be covered by a vesture, or the hand by a glove (not of smooth leather), for if the naked chest be struck by the naked hand a smack ensues which hides the character of the sound we wish to produce." [1] Corvisart percussed by the whole flat of the hand. At the present day immediate percussion is performed only upon the clavicles (they constituting pleximeters), or when the physician wishes to obtain a rough preliminary notion of the resonance of a considerable extent of the chest surface.

ii. *Mediate Percussion.*—Auenbrugger's glove was obviously an approach to that mediate percussion which was first systematically practised by Piorry.[2] Piorry interposes a thin plate of ivory (pleximeter) between the chest and the percussing fingers; the fingers moreover he keeps half bent, so that percussion is made by their very tips, and not by their pulps as in Auenbrugger's process. To substitute a hammer for the percussing fingers was not a great stretch of inventive genius: Laennec frequently percussed with his massive stethoscope: from time to time divers plessors have been contrived

[1] Inventum Novum, §§ 4, 5.

[2] Piorry. De la Percussion Médiate. Paris, 1828.

which may be seen in the shops of surgical instrument makers. But a hammer possesses no real advantage, and a finger of the left hand forms a perfect pleximeter: whence it comes that few physicians employ either plessor or pleximeter, except for the purpose of demonstration before a large class.[1] When the fingers are used to percuss with it is necessary that the movement of the hand should proceed from the wrist and not from the elbow or shoulder, a nicety in manipulation which is acquired by practice.

Article III.—Theory of Percussion.

The first object of percussion is to discover what kind of sound is emitted by the part percussed. A secondary object is to discover the degree of resistance, or the density of the stricken spot.

[1] Piorry. Traité de Plessimétrisme. Paris, 1866. Here the reader will find the argument stated in favour of the pleximeter. In the same book there is a description of sundry "plessigraphs" which have been contrived of late. I have employed M. Peter's plessigraph, and have not perceived its utility.

Wintrich: Einleitung zur Darstellung der Krankheiten der Respirationsorgane. Virchow's Handbuch, vi. I. Erlangen, 1854. Herein the cause of the hammer is pleaded.

¶ I.—PERCUSSION SOUNDS.

All sounds possess a certain loudness, pitch, and duration. Loudness is proportionate to the amplitude of the sonorous waves, to the distance over which they travel : pitch depends upon the number of undulations which occur within a certain space of time, the greater the number the higher the pitch : duration speaks for itself.

These three qualities being common to all sounds, there remains a property peculiar to some sounds, namely, tone. A tone, or musical sound, depends upon a rapid and rhythmical succession of equal sonorous undulations : in other words, a tone is produced by an exact repetition of sonorous shocks, which follow each other at regular intervals, with sufficient rapidity; not fewer than forty in the second. The pitch of a tone is high in proportion to the rapidity of this succession of waves. A noise, which is not a tone, depends upon an irregular succession of sonorous shocks, or upon the simultaneous production of several tones which do not harmonise.

The only tones, with which percussion has to do, are those which are produced by the vibra-

tion of stretched membrane.[1] Nor is stretched membrane capable of producing tones unless it vibrate over an air-containing cavity. Just as a vibrating string requires a sound-board, so does a membrane require a cavity, to take up and reinforce the vibrations. Under these conditions, namely, membrane vibrating over a cavity, another acoustic principle comes into play; resonance. The law of resonance is this: that columns of air of definite length resound to tones of definite rates of vibration. Experimentally it is found that the length of a column of air which most perfectly resounds to a tuning-fork is one-fourth of the length of the sonorous wave produced by the fork. This reinforcement of sound, this resonance, is due to the synchronism which is established between the vibrating period of the tuning-fork and that of the column of air.

In the case, previously supposed, of membrane stretched over a cavity containing air, when we strike the membrane we produce a flutter of the air contained in the cavity, and some pulse of this flutter which corresponds to the size of the cavity (according to the law just stated) is raised by resonance to the dignity of a tone. Which

[1] Bone and cartilage are excluded for the present.

tone is the same as that obtained when a tuning-fork, whose rate of vibration is that of the column of air contained in the cavity, is placed over the mouth of the cavity.[1]

I have now explained how it is that the words, tone and resonance, as applied to percussion-sounds, have come to be synonymous. The only tones, which percussion knows, are produced by resonance. But many percussion-sounds are not tones : and hence a fundamental distinction, the appreciation of which was, as we have seen, the first step made in the development of the art of percussion. The difference between resonance and non-resonance is indeed the difference between a tympanitic and an ascitic belly. Between musical tones and sounds which are wholly non-resonant, we meet in practice with all degrees of resonance. In proportion to the amount of tone possessed by any sound, does it become easy and necessary to estimate those three common properties before mentioned: to say that a sound is toneless suffices, there is no need to inquire further into its characters.

[1] See Tyndall's Lectures on Sound. London, 1867.

Class I.—Resonant Sounds.

I shall first consider resonant sounds with respect to their loudness, duration, and pitch; and afterwards with respect to the purity or clearness of their tone.

I. Loudness is not a character of much practical value, depending, as it does, not only upon the volume of the resonant material, but also upon the force of the percussion, and the clearness of the resonance.

II. Duration is a more important quality. The prime property assigned by Skoda to a percussion sound, its fullness or its scantiness,[1] (ideas adopted from Laennec,[2] and which Skoda himself confesses are not recognised by acoustics) seems to be, in reality, a composite conception, made up chiefly by the duration,[3] and partly by the loudness of the sound. Should a full sound be anywhere spoken of hereafter, a long loud resonance will be signified.

[1] The word "scanty" seems to me to be the best English equivalent of the word "leer" used by Skoda. Dr. Markham translates it, empty; but it is precisely empty things which give a "full" sound; "tinnit, inane est."

[2] Auscultation Médiate. 1826, i. page 28.

[3] Skoda implies as much; a full sound, he says, "appears more persistent."

III. Pitch, the last common property of sound, is practically found to be intimately associated with duration; certain variations in pitch being attended necessarily by certain variations in duration; the higher the pitch the shorter the duration, and the reverse. Arbitrary names have been given to a few resonances of the body according as they differ in pitch and duration.

Tympanitic resonance is the lowest and longest of all percussion-sounds; it is afforded by a tympanitic belly.[1]

Trachæal resonance is a phrase first used by Williams to signify the sound produced by percussion of the trachæa, the mouth being somewhat open. Trachæal (or tubular) resonance is much higher-pitched and shorter than the tympanitic.

Passing still higher upwards, as it were, we reach high-pitched sounds which are of very short duration, and therefore of very small reso-

[1] The English word, tympanitic, has a meaning very different from that of the German, tympanitisch. Tympanitic indicates a sound of a certain pitch and duration: tympanitisch refers to the coexistence of a "klang" or over-tone, segmental tone, an attribute present in many sounds not tympanitic. See Tyndall's Lectures for a full exposition of the subject of over-tones.

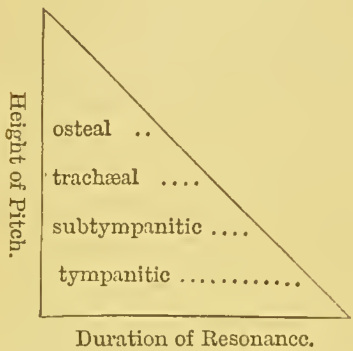
nance. These are the sounds afforded by the hard solid tissues, cartilage and bone, and hence called Osteal sounds by Piorry.

By raising the pitch still higher than it is in the osteal sound, the resonance becomes too short to be appreciable; we call the sounds produced, non-resonant.

But it is necessary to insert between the tympanitic and trachæal sounds at least one intermediate type of resonance; that is to say, a tone pitched higher than the tympanitic and lower than the trachæal, and of corresponding duration. This is the note afforded by percussion of the healthy lungs in their natural state of distension. And hence we might designate the resonance, pulmonary, but for this reason: the natural pulmonary resonance, although no doubt of the pitch and duration described, is not a clear resonance, but (as Auenbrugger pointed out) a muffled resonance: wherefore it seems best to reserve the epithet, pulmonary, for the muffled form. I propose to call the pulmonary resonance, regarded as to pitch and duration alone, Sub-tympanitic: which, like all resonances, may be clear or dull, qualities to be explained hereafter.

Precisely the same remark may be made re-

specting the epithet, tympanitic. The sound of a tympanitic belly is not a clear but a muffled resonance: yet it is convenient to retain the word tympanitic in the sense before defined, provided we indicate thereby merely the pitch and duration, and not the clearness of the resonance.[1]



So that we have a scale of resonances, wherein the pitch is progressively raised, and the duration progressively diminished, from the tympanitic to the osteal.

The Physical Conditions necessary to the production of perfect resonance (tympanitic, subtympanitic, or trachæal) are these: air contained within a sac of a certain kind. The sac may form a single cavity of considerable size, or may be subdivided into innumerable small sacs.

1st. Air contained within a single large sac.

[1] Stokes early drew this distinction: see Diseases of the Chest, Dublin, 1837, p. 335; where he speaks of a tympanitic clearness and a tympanitic dulness.

The sac may be completely closed, or partially open.

i. Sac completely closed: in this case it is necessary to the production of a perfect percussion-resonance that the walls of the sac be in a condition not only to generate the tone but also to transmit it. First: it is necessary to the generation of resonance that the internal surface of the sac be smooth, so as to be capable of reflecting the sonorous undulations of the contained air. Secondly: it is necessary that the walls of the sac transmit these sonorous undulations to the external air; that is, the sac itself must vibrate in unison with the contained air. And this unison-vibration of the walls of the sac requires that they be of a certain tension, neither quite relaxed nor excessively stretched: a fact which may be demonstrated upon the cheeks, and which is frequently demonstrated for us by the membrana tympani, a certain mean tension thereof being necessary to the perfect transmission of sound. Given these conditions of resonance in a closed sac, we shall find that the pitch of the sound depends upon the size of the sac, that is, upon the depth of the column of air percussed; the shorter this column, the higher the pitch of the percussion-

sound. Why this should be so, has been previously explained (page 65).

ii. Sac partially open. If a sac generate internal resonance, even though the walls transmit the sonorous undulations very imperfectly, we may yet hear a pure resonance, provided that the sac be partially open, so that the tone can pass out directly into the external air. This fact also may be demonstrated upon the cheeks: percuss one of them when quite relaxed, the mouth being shut, and scarcely any resonance is heard; open the mouth a little, and a pure sub-tympanitic resonance results. By opening the mouth wider and wider we can show that, the less perfect the sac, that is, the wider the opening by which the internal communicates with the external air, the higher is the pitch of the sound produced. In fact, it is necessary to the production of a tympanitic note in the human body that the sac be not open at all.

2nd. Air contained in innumerable minute sacs. We obtain these conditions in the lungs; but the lungs do not yield to percussion anything like a perfect resonance unless they are relaxed, such as is the case when they are removed from the body. Percussion of relaxed

lung yields a clear, and rather high-pitched (trachæal) resonance: we will discuss the cause of this fact hereafter. The presence of a certain amount of liquid in the air-cells of the lung, when it is in its state of natural distension within the thorax, will give rise to a similar resonance: the reason of this occurrence is not understood.

IV. Clearness. Having considered the varieties of perfectly resonant sounds we shall now see how that resonance may be impaired in its very essence of rhythmical and equal undulation. We have learned that by progressively raising the pitch of sounds we at length reach a height at which the duration of the sound is so short that all appreciable resonance ceases; the sound has become non-resonant by sheer lack in the quantity of sound. But non-resonance may be attained in quite another way: a tympanitic note (for example) may be made to become progressively less and less resonant, not by passing through the sub-tympanitic, trachæal, and osteal stages, and thereby diminishing the quantity of resonance, but by striking at the very quality of resonance itself (by disturbing the rhythm and equality of the vibrations), the tone becoming obscured, muffled,

damped, or dulled from the outset. This important distinction may be illustrated by reference to colour. Suppose a red patch having a certain area in square inches: the redness may be gradually washed off until none remains—impairment of quality; or the red patch may be diminished in size, inch by inch,—diminution of quantity, the redness, so long as any remains, being as red as ever. Or, again, the distinction may be manifested directly by an experiment in acoustics. Percuss the cheek as directed a short time back, and it will be found that the resonance may be not only diminished in quantity by opening the mouth widely, but also impaired in quality by increasing the thickness of solid material through which percussion is performed. This distinction was well known to Auenbrugger; and it is from him that we inherit the epithets of clearness and dulness.[1] A perfectly resonant sound is called a clear sound; as the resonance becomes more and more impaired, it is said to become less and less clear, until perfect dulness,

[1] "Sonitus vel altior, vel profundior (differencces in pitch and quantity of resonance), vel clarior, vel obscurior (differences in purity or quality of rcsonancc), vel quandoque prope suffocatus (perfect non-rcsonance)." Invent. Nov., § 10, Scholium.

tonelessness, or non-resonance, is reached. All the varieties of resonance which we have established according to the variations of pitch may possess perfect clearness, or may be more or less obscured or dulled. Only one form requires special notice, the muffled resonance yielded by the pulmonary regions of a healthy subject.

i. *Pulmonary Resonance.*—This was compared by Auenbrugger to the sound of a drum covered by a thick woollen fabric:[1] a muffled sub-tympanitic resonance. By experience alone can we learn what is to be reckoned a natural pulmonary resonance.

ii. *Other muffled Resonances.*—Obviously, there may be innumerable degrees of resonance between the clearest tones and perfect dulness. No special names are given to any of these sounds (except to the pulmonary sound just described): we simply use words which indicate that the resonance (whether tympanitic, sub-tympanitic, or trachæal) is impaired slightly, considerably, greatly, or is wholly absent.

The Physical Conditions necessary to the muffling or dulling of resonance are these.

[1] Inventum Novum, § 2.

i. Air contained in a single large closed sac, the walls of which are imperfectly vibratile. This diminution of vibratility is proportionate to either relaxation or distension beyond a certain point; that is, there is a certain degree of tension of the membranous walls which is accompanied by perfect resonance, any deviation therefrom diminishes the clearness of the sound. This fact also may be demonstrated upon the cheeks. That complete relaxation of the walls should diminish the resonance is easily understood, but why extreme distension of a resounding cavity should have the same effect (even up to the production of perfect dulness) is not quite so obvious. It cannot be that the walls are stretched too tight to vibrate, or that the contained air is too dense to vibrate: it seems as if the walls, arched by reason of their tension, bore off the percussion-vibration from the underlying air: depress a small part of the walls a little below the surface of the rest by means of a pleximeter (thereby to destroy the arch), and percussion upon the depressed pleximeter will give a clear sonorous note.

ii. Air contained in innumerable small sacs having tense and vibratile walls. This is the condition of the lungs during life. A short time

ago we saw that the lungs, when removed from the body, afford a clear resonance; now we learn that the lungs, when naturally distended, yield a muffled resonance. I do not profess quite to understand the reason of this. It may be that when the walls of the air-vesicles are relaxed their individual resonating power is reduced to a minimum, and the lung resounds as if it were a single sac. When the walls of the air-vesicles are distended their individual resonating power is retained; but, inasmuch as the innumerable stretched membranes do not vibrate in perfect unison (as indeed they do not, and cannot, receive the percussion impulse to vibration at precisely the same time), the purity or clearness of the resonance is diminished. A knowledge of these facts is so very important that a student should demonstrate them for himself upon the dead body (of a child, if possible). Let him percuss the chest and note the pitch and clearness of the resonance; then let him remove one lung, and percuss it when it has become retracted, and he will find that the note is very much clearer than before, although the pitch is higher. The heightening of pitch in the shrunken lung is due to diminution of the column of air percussed. Upon the other side

of the chest another instructive experiment may be performed. Let the chest be percussed, then let a small trocar and cannula be passed into the pleura, and air be injected through the cannula, but so as not to distend the side in the least. It will be found that the percussion sound is not materially changed from what it was before the chest was opened. Now let air be injected so as to distend the pleura gradually to the utmost; and it will be observed that the percussion note gradually gains in duration and clearness of resonance up to a certain point, and falls in pitch.

iii. The presence of non-resonant material which deadens or muffles the vibration of the percussion blow and of the resonant part percussed, will proportionally obscure the resulting resonance. For instance, in the case of the chest, increase in the thickness of the thoracic parietes will diminish the clearness of the percussion note to any degree between the natural pulmonary resonance and absolute dulness.[1]

[1] See Table, opposite page.

		Physical Conditions.		Resonance.	Pitch.
		Walls of Cavities.	Column of Air.		
Lung	shrunken	Innumerable: but possessing no individual capacity of vibration: cavity virtually single.	small	clear ..	high.
	distended	Innumerable: possessing individual capacity of vibration, but not in unison.	considerable	muffled	medium
Pneumothorax	not distended	Single: but relaxed, and incapable of perfect vibration.			
	distended	Single: tense and capable of perfect vibration.	great	clear ..	low.

Class II.—Non-resonant Sounds.

We have seen how non-resonance is approached by impairing the quality or diminishing the quantity of resonance. The pitch and duration of a non-resonant sound are difficult to

appreciate, and may be wholly disregarded, except when comparison is actually made between the amount of non-resonance of different bodies.

The Physical Conditions of non-resonance are these:—

i. All soft animal tissues (except stretched membrane) and all liquids are non-resonant.

ii. Superficial percussion of an air-containing cavity with greatly distended walls, will, as before said, evoke a dull sound.

APPENDIX.

Metallic Sounds.

Certain by-sounds, which are called Metallic, sometimes accompany the sounds which have been described. Metallic sounds are two: amphoric and cracked-pot.

1st. Amphoric resonance is the product of an over-tone superadded to the fundamental tone; it is the tympanitic quality of Skoda carried to the highest degree possible in the human body. Fillipping the cheeks, blown out to their very fullest, affords an amphoric note. The Physical Conditions necessary to metallic resonance are illustrated by this experiment; namely, a large cavity, with very tense walls, containing air.

Hereafter we shall learn that auscultation, under the same circumstances, detects phenomena of precisely the same kind; the amphoric echo, metallic tinkling, and metallic ringing.

2nd. Cracked-pot sound was discovered by Laennec.[1] It is due to the sharp escape of air through a narrow chink or hole, and may be imitated by clasping the hands loosely together, and striking the back of one of them upon the knee. The Physical Conditions necessary to the production of a cracked-pot sound are these:—that a certain amount of tissue, containing air which communicates with the external air, be enclosed between two solid opposed surfaces; and that neither the amount of contained elastic aërated tissue, nor the resistance of the peripheral hard walls, be so great as to prevent the percussion blow from forcing out a little of the air. Whence we easily understand why, when the chest-walls are yielding (as in children) perfectly healthy lungs should sometimes give a cracked-pot sound; and why, when the chest-walls are resisting (as in adults) a cracked-pot sound mostly indicates the presence of air-containing tissue or cavity,

[1] Ausc. Méd., i. pp. 100, 655.

interposed between the chest-wall percussed, and solidified lung or a pleural effusion behind. A firm blow, the state of expiration, and open mouth and nostrils, are very conducive to the production of a cracked-pot sound.

A sound practically indistinguishable from the cracked-pot sound is that to which Piorry has given the name of "metallic," and which is sometimes produced by percussion over cavities which contain both liquid and air. The sound in question is a sort of percussion-rale due to splashing of the liquid in the cavity.

¶ II.—PERCUSSION RESISTANCE.

The sense of resistance perceived by the percussing fingers is greater or less in proportion to the greater or less compressibility of the part percussed. Hence solids and liquids are very resistant, air-containing parts much less so. We should always appreciate the amount of resistance; it is by this means that the person percussing always learns much more from his percussion than does a mere bystander. Corvisart, in his notes to Auenbrugger, first drew attention to these signs: Piorry, working out this idea, has at last almost come to exalt the tactile sensations of percussion above the acous-

tic. But, apart from all exaggeration, there is no doubt that by the sense of resistance we can distinguish between certain states which otherwise agree in affording absolute dulness to percussion. The excessive resistance of a liquid effusion, and the change which we so often perceive on passing from the heart to the liver, are examples of this fact.

Before I proceed to apply these principles to the practice of the percussion of the chest in health and disease, it will be proper to say a few words upon what are called Superficial and Deep percussion. By progressively increasing the force of the blow from the gentlest tap to the hardest the patient can bear, we influence progressively deeper layers of the part percussed. For example: gentle percussion will elicit as good a pulmonary resonance an inch or two below the right nipple as above the nipple; but hard heavy percussion will produce a note which is much less full in the former than in the latter spot: the true explanation being this; not that deep percussion brings out hepatic non-resonance below the nipple; but that, while gentle percussion influences only a small depth of lung, which depth exists as well below the nipple as above it, deep percussion, in-

fluencing a much greater depth of lung, shows, by a marked difference in the fulness of the sounds, that the necessary thickness of lung does not exist over the liver, but does exist above it.

In like manner the direction of the blow modifies the sound produced: different parts being influenced according as the blow is quite perpendicular to the surface, or oblique, or nearly horizontal. As a rule the blow should be struck quite perpendicularly.

SECTION II.

PERCUSSION OF THE CHEST IN HEALTH.

The pulmonary, cardiac, and mediastinal regions must be separately considered.

Article I.—The Pulmonary Region.

¶ I.—PULMONARY RESONANCE AND RESISTANCE.

i. *Typical.*—Frequent experiment upon the healthy chest is the means by which to fix in the mind an idea of the sound and resistance afforded by percussion of the pulmonary regions. When disease affects one side only we possess in the other side a standard of health to which we

may refer: even when both lungs are affected, but one more than the other, comparison is still useful: wherefore, to contrast the same parts of the two sides of the chest becomes an important rule in the practice of percussion.

ii. *Sub-typical.*—But certain deviations from the type are compatible with a state of perfect health; deviations for the most part due to the thoracic walls. The sound produced by percussion over the sternum, clavicles, ribs, and scapular spines, partakes of the osteal character. Ossification of the cartilages produces the same effect. The greater the quantity of soft tissue, muscular or adipose, which covers the thorax, the greater the muffling of the sub-tympanitic resonance. Hence the percussion-note of the chest is clearer in front, and at the sides, than behind; clearer in thin persons than in fat; it is sometimes almost impossible to get any sound deserving the name of resonant by percussing the backs of fat flabby people. When the chest-walls are yielding, heavy percussion of the front of the chest will produce the cracked-pot sound. The resonance is much more clear in children than in older persons. Wintrich has shown that, during the long deep expiration which attends screaming, the chest of children becomes

very much less resonant than natural; a fact which it is most important to remember.

¶ II.—EXTENT OF PULMONARY REGION.

The region which yields a pulmonary note extends from the very apex of the thorax on each side, as low as the sixth rib in front, the seventh at the sides, and the tenth or eleventh behind. But sundry viscera encroach upon these limits.

(i.) The Heart causes a certain extent of non-resonance in the anterior part of the chest; see next page. (ii.) The Liver can, by hard percussion, be detected on the right side, as high as the fifth or even the fourth intercostal space in front, and ninth or tenth rib behind. (iii.) The Spleen on the left half of the chest, below the sixth rib laterally, modifies the percussion sound. (iv.) The Stomach, especially when distended with gas, affords its own resonance to percussion of the lower part of the left side of the thorax, as high, it may be, as the fifth rib.

Article II.—The Cardiac Region.

The extent of cardiac percussion dulness will differ according to the force used in percussion,

whether slight or great. Gentle percussion detects dulness only where the heart is uncovered by lung; this is the area of superficial dulness. Stronger percussion detects the dulness of the heart where it lies behind the lung; this is the area of deep dulness. The difference depends, as before explained, upon the mass of lung influenced by percussion.

i. The Superficial area is roughly triangular in shape: the right side of the triangle being the midsternal line from the level of the fourth chondrosternal articulation downwards; the hypotenuse being a line drawn from the same articulation to a point immediately above the apex-beat; the base being a line drawn from immediately below the apex-beat to the point of meeting between the upper limit of liver dulness and the midsternal line. The area of superficial dulness is much diminished by a deep inspiration; much increased by the patient lying upon the left side (the same position which displaces the impulse to the left); and not materially affected by the patient lying upon the right side.

ii. The Deep-seated area reaches upwards as high as the third rib (in children even as high as the second interspace); to the left about a finger's

breadth to the left of the impulse; and to the right as far as a little beyond the right margin of the sternum.[1] But, in truth, the right limit of cardiac dulness is not very trustworthy; the osteal and conducted pulmonary notes interfering much with the cardiac percussion sound.

No doubt it is sometimes quite easy to be able to discover the lower margin of the heart by percussion: sometimes a heightening of pitch and increase of resistance are tolerably well marked on passing from the heart to the liver; sometimes there is a distinct band of faint resonance between the two organs; and sometimes, in passing from the hepatic to the cardiac region, one becomes sensible of a slight increase in the intensity of dulness, and a most distinct increase in resistance; owing probably to comparative thinness of the left lobe of the liver, and its position over the stomach. When the heart and liver dulnesses pass indistinguishably into each other, we must be content with assuming the lower margin of the heart to correspond with a line drawn from a little below the apex-beat of the heart to the point of meeting between the right limit of cardiac dulness and

[1] Kobelt: Ueber Form und Dimensionen der Herzdämpfung. Arch. für Heilk., 1863.

the upper limit of hepatic dulness; bearing in mind how intimate is the connection between the heart and liver by means of the vena cava.

Article III.—The Mediastinal Region.

The contents of the mediastinum in the healthy state do not affect the percussion-note in any way.

SECTION III.

PERCUSSION OF THE CHEST IN DISEASE.

Article I.—The Pulmonary Region.

Auenbrugger's Dictum.—If, over the fore-described pulmonary region, there is not perceived the fore-described pulmonary sound; equal on both sides, the force of percussion being equal; we may predicate the existence of disease where the sound is preternatural.[1] This is Auenbrugger's dictum, and comprises the whole theory of percussion.

[1] "Si igitur ex prædictis locis sonoris non percipitur sonus manifcstus, utrique latcri æqualis, eidem percussionis intensitati conformis, morbosum quid in pectore latere significat." Invent. Nov. § 11.

¶ I.—PULMONARY RESONANCE.

The typical pulmonary note may be deviated from by increase or diminution of its resonance. Hence two classes of unnatural sounds.

In the following passages it is assumed that the chest walls are elastic and not too fat: in practice it must ever be borne in mind that the more the rigidity and fatness of the chest walls are increased, the more is the resonance afforded by percussion of them diminished.

Class I.—Increased Resonances.

The resonance is increased by the note becoming tympanitic or clear. In Skoda's phraseology we should say that the note becomes fuller (and not more muffled) or clear: the fulness of a sound being closely bound up with its duration, which is increased when a pulmonary note becomes tympanitic.

1stly. Tympanitic Resonance, mostly muffled, but occasionally clear, occurs when the sonorous column of air is greater than natural; a condition present in pneumothorax and in emphysema.

2ndly. Clear Resonance, sub-tympanitic or trachæal, occurs when the conditions which

render the natural pulmonary sound muffled, are removed; no new causes tending to diminish resonance being introduced. Clear resonance is always limited in extent, is never heard over the whole of one side of the chest. The circumstances under which we meet with clear resonance are these :—

i. Relaxed lung tissue, when in contact with the chest wall, affords at that spot a clear sub-tympanitic or trachæal note. Pleural effusions always relax the lung; and so sometimes do pulmonary consolidations, and tumours or enlarged organs without the lung.

ii. Cavities, filled wholly or chiefly with air, when at the surface of the lung, or when, if deep-seated, separated from the surface by dense solid tissue, afford a clear sub-tympanitic or trachæal resonance. Cavities surrounded by natural lung tissue, do not materially affect the percussion note.

iii. Solid masses, coming to the surface, and closely connected with large air-passages, sometimes yield a pure trachæal resonance: pneumonia and pulmonary tumours occasionally illustrate this fact.

iv. Liquid or solid, intimately mingled with aërated tissue, may cause a clear resonance, sub-

tympanitic or more often trachæal. Hence the clear sounds sometimes afforded by catarrh, œdema, congestion of the lung, pneumonia at its very outset or during resolution, phthisis, and tubercle.

Class II.—Diminished Resonances.

The resonance is diminished by the note becoming short and high-pitched, or more muffled. In Skoda's phraseology, the sound becomes scanty or more muffled. When absolute non-resonance is present, it is vain to draw (so far as the sound is concerned) any further distinctions. When a certain amount of resonance is preserved, it is not only possible but useful to distinguish between the diminished resonance which is due to diminished duration of sound, and that which is due to increased muffling. The following morbid states bring loss of resonance about:—

i. Liquid or solid exudations into the lung, whereby the amount of resonant material is diminished, raise the pitch and lessen the duration of the percussion-note in all degrees up to complete absence of resonance. Catarrh, œdema, congestion, pneumonia, phthisis, afford illustrations daily of this fact.

ii. Exudations into the pleura, whereby the lung is separated from the chest-walls, muffle the resonance in all degrees up to absolute dulness : a fact exemplified by liquid effusions, and by old adhesions.

iii. Extreme distension of the lung with air, whereby the chest-walls become so arched as to bear off the percussion-blow from the parts beneath, is attended by all degrees of impairment of resonance.

APPENDIX.

METALLIC SOUNDS.

I. Amphoric resonance occurs in pneumothorax, and in enormous pulmonary cavities containing air.

II. Cracked-pot sound, when a morbid phenomenon, is produced by percussion over a small superficial cavity, or over a thin superficial layer of healthy lung, backed by dense consolidation of the tissue behind, or by a liquid effusion.

¶ II.—PULMONARY RESISTANCE.

Like resonance, so the sensation of resistance perceived by percussion depends very much

upon the character of the chest walls. The resistance is increased by whatever diminishes the elasticity of the part percussed. Wherefore massive consolidations of the lung, large pleural effusions, and excessive distension of the lung or pleura with air, afford a sense of great resistance.

¶ III.—EXTENT OF PULMONARY REGION.

Over the præcordial region, percussion is employed in order that we may learn whether the lung has shrunken away from the heart, or covers it, or whether the pleura contains air. In the diagnosis of adherent pleura, of phthisis, or of emphysema of the left lung these signs become useful.

The discovery of the position of the diaphragm and mediastinum, and of contraction of the pulmonary apices, depends chiefly upon percussion. These important subjects will be discussed hereafter.

Article II.—The Cardiac Region.

I have just shown how diseases of the lungs or of the pleuræ influence the area of cardiac dulness; I will now proceed to the effects of disease of the heart itself. The area of dulness

(superficial and deep) may be diminished or increased.

i. *Area diminished.*—This occurs in the very rare cases of pneumopericardium, clear resonance superseding the natural dulness: a cracked-pot sound has even been heard in such cases. Atrophy of the heart does not materially affect the area of dulness.

ii. *Area increased.*—This occurs in enlargement of the heart, and in pericardial effusion: for the diagnosis between these conditions the reader is referred to the second part.

Moreover the area of dulness may be altogether displaced, in consequence of displacement of the heart; a subject considered in the chapter upon inspection (page 39).

Article III.—The Mediastina.

Dilatation of the large vessels, aneurysms, and solid tumours are sometimes the cause of more or less dulness to percussion where there should be resonance; as will be further shown in the special part.

CHAPTER V.

AUSCULTATION.

SECTION II.—AUSCULTATION OF THE LUNGS AND PLEURÆ.

Art. II. Respiratory Sounds—*continued :*

¶ II. Respiratory Sounds in Health.

¶ III. Respiratory Sounds in Disease.

i. Weak Breathing.

ii. Loud Breathing.

iii. Bronchial Breathing.

Appendix : Rales.

Art. III. Pleural Sounds :

¶ I. Friction Sounds.

¶ II. Amphoric Sounds :

i. Amphoric Echo.

ii. Metallic Tinkling.

iii. Bell-sound.

iv. Splashing Sound.

Physical Conditions of amphoric Sounds.

SECTION III.—AUSCULTATION OF THE HEART.

Art. I. Endocardial Sounds :

¶ I. In Health :

Causes of the Sounds.

Loudness of the Sounds.

Reduplication of the Sounds.

¶ II. In Disease ; Murmurs :

i. Place of Murmurs.

ii. Time of Murmurs.

iii. Diagnostic Signification of Murmurs.

Art. II. Pericardial Sounds :

¶ I. Pericardial Friction.

¶ II. Splashing Sound.

Appendix : Sounds, neither endo- nor pericardial, produced by the movement of the heart.

SECTION I.

INTRODUCTORY.

Article I.—Historical.

AUSCULTATION existed not before Laennec. True, a few passages are found in the writings of earlier physicians[1] which speak of sounds heard in the chest, but the observations remained mere curiosities, wholly without influence upon practical medicine. It is interesting to trace the dawning of the great discovery. Corvisart had studied the different forms of

[1] The following are the chief notices of auscultation contained in authors prior to Laennec :—

i. Leather sound of pleural friction. Hippoc. de Morbis : ii. § 57 ; ibid. iii. § 7.

ii. Friction-rale ? Hippoc. de Morbis : ii. § 59. See Van Swieten : Comment. in Aphor. 1219.

iii. Succussion-splash. Hippoc. de Morbis : i. § 5 ; ii. § 45 ; iii. § 26. § 27. De Intern. Affect. § 24. Coacæ Prænot. 432.

iv. Heart's sounds. Harvey : de Motu cordis et sanguinis, cap. v.

v. Pneumonic crepitation. Van Swieten : Comment. in Aphor. 826. Josephi Quarin : Methodus medend. inflamm. Vindob. 1774, p. 101.

vi. Respiration bruissante. Double : Séméïologie, ii. p. 31, 1817.

vii. Sounds of foetal heart. Mayor : quoted by Laennec, ii. p. 459.

enlargement of the heart, and had endeavoured to distinguish between active and passive enlargement; to this end the character of the impulse was carefully observed. Bayle, a disciple of Corvisart, was in the habit of applying to the heart-region his ear rather than his hand,[1] inasmuch as a heaving impulse is more readily detected thereby, be the reason what it may. Laennec, Bayle's friend and fellow-student, adopted the same method. Laennec had undergone a fifteen years' training in the hospitals of Paris when, in 1816, he was consulted "by a young person who presented the general symptoms of disease of the heart, and in whom palpation and percussion gave no information, on account of the patient's fatness. Her age and sex forbade an examination of the kind just mentioned (by putting the head to the chest), when I remembered a well-known acoustic fact, that if the ear be applied to one end of a plank, it is easy to hear a pin's scratching at the other. I conceived the possibility of employing

[1] "I have found this method nowhere alluded to, and Bayle was the first whom I saw employ it, when we followed the practice of Corvisart together. The professor himself never put his head to the chest." Ausc. Méd., 2me édit., i. p. 5.

this property of matter in the present case. I took a quire of paper, I rolled it very tight, and applied one end of the roll to the præcordial region; then leaning my ear on the other end, I was surprised and pleased to hear the beating of the heart much more clearly than if I had applied my ear directly to the chest."[1] He had discovered auscultation. At the Hôpital Necker he explored the new world gained for science; and in 1818 he read a 'Memoir upon Auscultation by divers acoustic instruments, employed as means of investigation in the diseases of the thoracic viscera, and especially in pulmonary Phthisis.'

In 1819 he published the first edition of his book on Mediate Auscultation; and, in 1826, the second.

Article II.—Methods of Auscultation.

It was by means of an instrument that Laennec was enabled to discover the powers of Auscultation. Since his day the stethoscope has been discarded by many persons, who, preferring immediate to mediate auscultation, apply the ear directly to the chest. However the

[1] Ausc. Méd., 1re édit. i. p. 7.

drawbacks to immediate auscultation are great, and chiefly these: the impossibility of listening to every patient's chest without the interposition of some kind of vestment, which, besides being a bad conductor of sound, gives rise to divers noises of its own; the impossibility of applying the ear to every portion of the chest; the impossibility of localising sounds with sufficient accuracy. No doubt a person may become a skilful auscultator who listens with his unassisted ear: but let mediate auscultation ever be considered the rule of practice, and immediate the exception: the physician making of the stethoscope, not a crutch, but a staff, which he uses when he has it, yet when he has it not he does not want it.

The stethoscope should be turned out of light wood; exact shape and size thereof quite unimportant: the student ought to fix his attention upon the training of his ear, and disregard the fashion of his instrument. Yet inasmuch as the shape of the stethoscope somewhat influences the character of the sounds heard, it is necessary to say that the use of Piorry's modification of Laennec's stethoscope (that commonly employed in England) is presupposed throughout the ensuing pages.

SECTION II.

AUSCULTATION OF THE LUNGS AND PLEURÆ.

Auscultation of the surface of the chest is practised with reference to three kinds of sounds: i. the Voice, as it is heard over the thorax: ii. the sounds of Breathing: and iii. the sounds produced in the Pleura.

Article I.—Thoracic Vocal Sounds.

The voice, as it is heard upon the surface of the chest, is usually called the Vocal Resonance: a phrase which we inherit from Laennec, and which involves a theory not yet discussed. It is useful to study the vocal resonance before the respiratory sounds; inasmuch as the origin of the sound is, in the former case, clear and indisputable, but, in the latter case, not so. Let us follow the historical order: the first definite result yielded by auscultation concerned the vocal resonance.

¶ I. THEORY OF VOCAL RESONANCE.

The previous researches into the theory of percussion sounds will be found very much to have cleared the way into the present subject.

The voice is a tone: what we have to consider is the manner in which that tone is modified by the time it reaches the surface of the chest. And the prime distinction between vocal resonances (just as between percussion-sounds) depends upon the clearness of tone. Whence two classes of vocal resonances; the muffled, and the clear. Clear vocal resonance, being usually heard over the large air-tubes, is also called bronchophony.

i. *Muffled Vocal Resonance.*—The ear, applied to the chest of a person who is speaking, perceives an indistinct humming noise: the laryngeal tones have lost their clearness, or definite musical character. Their loudness also is diminished. Their pitch is manifested chiefly in the influence it exerts over the loudness of the thoracic voice; the deeper the voice, the greater the vocal resonance; just as palpable vocal fremitus is strong in proportion to the depth of the voice.

ii. *Clear Vocal Resonance, or Bronchophony.*—What vocal resonances shall be deemed clear enough to form a class apart from the muffled vocal resonances is obviously an arbitrary arrangement. The line is artificially drawn at that amount of clearness which the voice pos-

sesses when listened to in the upper part of the interscapular regions, in a certain proportion of perfectly healthy persons. Taking a number of persons (age immaterial) we auscult them, while speaking, at the spot indicated: in some we hear nothing different from what we hear over the rest of the chest; but in some we hear a vocal resonance which is comparatively clear, that is to say, which approaches more nearly in character to the tones heard over the larynx. Bronchophony then I define to include the clearest vocal resonance ever heard over the healthy chest, and all degrees of clearness greater than this. For disease sometimes affords thoracic vocal tones which are far clearer than any heard in health; tones which are not inferior to those heard over the larynx. And, just as the least clear vocal resonance, which can be called bronchophonic, is that which is heard over the healthy bronchi; so does auscultation of the larynx afford a standard of the most clear vocal resonance. For it is not possible (generally speaking) that the voice should ever be heard over the chest, in health or in disease, with greater loudness or clearness than over the larynx.

The clearness of vocal resonance is quite in-

dependent of its loudness; a whisper may be as purely bronchophonic as the loudest sounds: and, in practice, when bronchophony is very clear, we set aside as far as possible the element of loudness, in order the better to appreciate the degree of clearness; we bid the patient speak in a whisper, else, should he speak aloud, the thoracic sound would become so great as to cause pain to the examiner, and to destroy all further estimation of the properties of the sound. Bronchophony is sometimes brought to a close by a very well-marked bronchial expiratory sound.

Ægophony.—A curious variety of bronchophony is that which was called, by Laennec, ægophony, in order to indicate its resemblance to the bleating of a goat. Still closer is the comparison which he makes between ægophony and the voice of Punch. Bronchophony may become ægophonic in any degree between the merest trace of tremulousness, and a nasal twang of the most remarkable kind.

Pectoriloquy:—Is the name which Laennec gave to that physical sign which was the subject of his earliest publication relative to auscultation. Pectoriloquy having thus been the first-fruits of his discovery, no wonder that he always

clung with a feeling of affection to the name and to the object signified. Laennec meant by pectoriloquy, an apparent transmission of the vocal resonance along the canal of the stethoscope to the ear of the observer: a phænomenon of the same kind as the souffle or puff, which sometimes attends the breathing-sound. The criterion of pectoriloquy consists not in the clearness of the vocal resonance; nor in its loudness, since a whisper may be conveyed along the stethoscope as perfectly as the most insupportable bronchophony.

The vocal resonance, though useful chiefly as a confirmation of the notions acquired by auscultation of the respiratory sound, sometimes acquires an independent value. When a patient breathes so as scarcely to expand some parts of his chest, or breathes noisily, or otherwise unnaturally, then we call in the aid of the vocal resonance to remove our difficulties.

Physical Conditions of Vocal Resonance.

The vocal resonance has been defined to signify the sound produced in the larynx and heard upon the surface of the chest. Wherefore two points demand examination: What

sound is produced in the larynx? How does that sound reach the surface of the chest?

The larynx is a reed instrument which is capable of sounding a fundamental tone. Sundry minor or segmental tones, overtones, are produced along with the chief tone. The vocal chords may vary the pitch of the tone they produce, but no change in the essential quality of the sound can be effected in the larynx itself. The mouth, however, by varying its shape, can be made to resound to any of the laryngeal tones; and it is to different admixtures of these tones, occurring in the mouth, that the different vowel sounds are due. Consonants obviously are produced by the mouth and nose. Wherefore the larynx has nothing to do with articulation: the laryngeal sound, whatever that may be, is one sound. Perhaps, of all the vowels, the vowel āh approaches nearest to the fundamental laryngeal tone: at any rate the mouth and fauces seem to be most quiescent during the production of that sound.

The next consideration concerns the manner in which the laryngeal tone (say the vowel ah) reaches the surface of the chest. The sonorous undulations pass upwards and downwards from the vocal chords; and just as the pharyngeal

vault, the nasal fossæ, and the mouth play the part of an arched roof, so does the trachæa that of a speaking-trumpet. The inner surface of the windpipe reflects the vocal vibrations which would otherwise diverge, and confines them in the tube, so that the voice is carried in all its fullness down the windpipe; and would be heard at the bifurcation of the trachæa, as well as at the vocal chords, were it not for one circumstance, namely, that the current of air is in a direction reverse to the propagation of the sound. Hence one cause of weakness of the thoracic voice to begin with.

But the analogy of the speaking-trumpet ceases at the bifurcation of the trachæa. The progressive diminution in the size of the air-tubes, and in the rigidity of their walls, renders the reflected sound progressively less in amount; at the same time that the multiplication of soniferous columns of air, differing in length, renders the tone less clear, because its constituent fractions are not in unison. Wherefore the voice, heard over the surface of the lungs, has lost both in loudness and in clearness, is both weak and muffled. When we compare the single sectional area of the windpipe with the enormous number of minute air-tubes which

terminate at the surface of the lung, it is not difficult to conceive how the laryngeal voice becomes frittered away as it passes onwards.

But why is the bronchophony of the larger tubes not heard above the vesicular vocal resonances? Because the pulmonary tissue in its naturally distended state, consisting of an incessant alternation of air and membranous walls (themselves capable of individual resonance), is a bad conductor of sound, whether as to loudness or to purity of tone: and the bronchophony must pass straight through the lung; the air vesicles cannot accommodate a sound of a hundred times less volume. It is hardly necessary to prove that distended lung does conduct sound badly: percussion already has shown that it does so; and let a person who has any lingering doubts upon the point explain, why the heart-sounds are so much weakened by passage through emphysematous lung, and why bronchophony often ceases to be heard the instant the stethoscope passes from off the trachæa on to the pulmonary regions.

Hitherto I have considered vocal resonance as if it were altogether a conducted sound. Now, by conduction, a sound may lose in loudness and clearness, but cannot gain therein.

Wherefore simple conduction is obviously incapable of explaining those cases (comparatively unimportant) in which the voice becomes actually louder, or longer, or higher, or altered in quality, by transmission through the lungs. Increased loudness or length of vocal tone, heard over a cavity at the surface of the lung, may be explained by resonance. The changes in quality are chiefly two; ægophony, and the sniffing bronchophony of pneumonia. Ægophony is probably due to flattening (not amounting to complete collapse) of the smaller air-tubes, which, during the bronchophony, undergo incessant changes in caliber. The sniffing quality of pneumonic vocal resonance seems to be caused by an admixture of bronchial (or tubular) expiratory sound. To conclude: excepting the cases just referred to, those in which the loudness of the voice is reinforced in the chest, it really does appear that resonance (in the acoustic sense) has little or nothing to do with the production of ordinary vocal resonances; that in fact the phrase, vocal resonance, can be continued in use only under the distinct understanding that the substantive is employed in a sense quite peculiar to the nomenclature of auscultation.

¶ II.—VOCAL RESONANCE IN HEALTH.

The vocal resonance over the pulmonary regions is muffled. Behind, opposite the trachæa and the bifurcation of the bronchi (over the upper dorsal spines), bronchophony is heard in a certain proportion of healthy persons: a circumstance probably accounted for by the fact that only solids intervene between the windpipe and the surface of the body at the spot indicated. Under the clavicles, near the sternum, the vocal resonance (especially on the right side, and in women) is often bronchophonic: the right bronchus is larger and lies higher than the left; and in women, the low position of the shoulders renders the position of the bronchi relatively higher than in men. The voice is naturally more laryngeal and sonorous in some persons than in others: it is, in women and children, oftentimes inaudible over a large portion of the chest.

¶ III.—VOCAL RESONANCE IN DISEASE.

i. *Increased Clearness of Resonance: accidental Bronchophony.*—Whatever increases the conducting power of the lung increases the clearness of the vocal resonance. The conducting

power of the lung is increased by whatever diminishes the heterogeneity of its structure. Diminution of the quantity of air contained in a part of the lung, diminution of the number of the alveolar septa; either of these changes will be attended by increased homogeneity. For these reasons it will be apparent why consolidation of the lung and cavities are the two chief causes of bronchophony. Consolidation of the lung includes simple collapse, hæmorrhagic infarctions, and new formations (pneumonic, phthisical, cancerous). Cavities are due to phthisis or dilatation of the bronchi. Occasional causes of bronchophony will be found in œdema of the lungs (with comitant hydrothorax) and emphysema.

ii. Diminished clearness of resonance is an unimportant sign, except when it is known to have supervened upon resonance præternaturally clear. And, in such cases, the soniferous power of the bronchi is at fault, in consequence of collapse, or of obstruction by mucus, blood, or exudation.

iii. *Ægophony and Pectoriloquy.* — It is no longer possible to credit ægophony with the definite value attributed to it by Laennec; namely, that it indicates the presence of a thin

layer of fluid in the pleura. Superficial collapse of the lung (whether due to liquid effusion or to false membranes) is all that is necessary to produce perfect ægophony.[1] Ægophony, when well-marked, is mostly heard about the angle of the scapula. Nor has pectoriloquy retained its early reputation for being an infallible sign of a phthisical cavity. Tumours of the lung, consolidation, simple collapse, may each and all be attended by perfect pectoriloquy. Nevertheless, I believe that Laennec was right in supposing that pectoriloquy does mostly indicate that the seat of the bronchophony is near the surface.

Resonance of the Cough and Cry. In infants the thoracic resonance of the cry sometimes affords useful evidence of disease; for instance, the cry heard through hepatised lung is strongly bronchophonic. In like manner, the resonance of the cough sometimes acquires value when other signs are absent. Whatever has been said of the voice applies to the cough and cry.

[1] Landouzy: De la valeur de l'Egophonie. Arch. de Méd., 1861.

Article II.—Respiratory Sounds.

¶ I. THEORY OF RESPIRATORY SOUNDS.

The fundamental division of respiratory sounds is the same as that of vocal resonances. The respiratory sound, which is heard over the larger air-tubes, differs by the possession of a certain quality, from the sound which is produced in the spongy structure of the lung. Wherefore the former is called Bronchial, and the latter Vesicular breathing.

i. *Vesicular Breathing.*—The ear, applied to the breathing chest, detects a sound which can be defined only by the negative property of not possessing the bronchial quality. Wherever breathing lung is in contact with the chest-wall, there we hear this sound. Its inspiratory portion has a duration equal to that of the inspiratory movement; the expiratory portion follows without any interval, has a duration only one-fourth or one-third of that of the inspiratory sound, and is much less loud than it.

The sound is loud in proportion to the rapidity and depth of the breathing. The louder the sound the greater becomes the relative duration of the expiratory portion; and for this reason;

the expiration-sound being potentially equal in duration to the expiration-movement (although during the latter part of the movement the sound is habitually inaudible by the sharpest ear), whatever quickens and deepens the expiration-movement makes the whole sound louder, and the audible sound longer. Sometimes the tranquil breathing of adults is unattended by expiratory sound. Sometimes, especially in fat middle-aged women, the breath sound can be hardly heard at all. Loud vesicular breathing was called Puerile by Laennec.

Sometimes the inspiratory sound, instead of being continuous, is divided into three or four distinct parts: occasionally the expiratory sound likewise is duplicate: this is called jerking breathing: a sign of no practical importance.[1]

Sometimes the chest is felt to heave with inspiration before any sound is heard: this has been called deferred inspiration.

Sometimes the expiratory movement is prolonged in consequence of obstruction to expiration; when this is the case, the sound is prolonged also.

ii. *Bronchial Breathing.*—Bronchial breathing,

[1] See Roger: L'Union Méd. 1861, No. 120.

like bronchophony, is heard about the upper dorsal vertebræ in a certain number of perfectly healthy persons. Bronchial breathing is distinguished from vesicular breathing by the possession of a special quality of sound. Rightly to understand that bronchial breathing is not determined by the loudness, pitch, or duration of the sound, inspiratory or expiratory; and to fix in the mind a true idea of what that quality is which we call bronchial, constitute the most important part of the doctrine of pulmonary auscultation. This bronchial quality is far from being the same in all cases: that is to say, there are different kinds of bronchial breathing. A person, who had become familiar with that special sound-quality alone which respiration produces in phthisical cavities, would be exceedingly amazed, when he heard for the first time the breath-sounds of hepatised lung, and was told that they also are bronchial. In fact, as has been already said, there are divers kinds of bronchial breathing.

Laennec set the example of denoting the different kinds of bronchial breathing by special names. For my own part, I agree with those who maintain that the present state of physical

diagnosis does not justify the breaking-up of bronchial breathing into varieties. Different though the quality and physical causes of bronchial breathing may be in different cases, yet no one hitherto has been able to connect any special variety of bronchial breathing with a constantly corresponding condition of the lung. For example: the existence of a cavity in the pulmonary tissue undoubtedly introduces a new element into whatever respiratory sounds are heard over that cavity; nevertheless breathing, identical in all respects with that which Laennec designated cavernous, is sometimes heard when nothing like a cavity is present. However, I shall point out the sense in which a few such words, frequently employed, are used.

It is bronchial breathing which is heard in many healthy persons between the shoulder-blades. This circumstance affords an excellent means of studying one special bronchial quality. It may be roughly imitated by blowing gently through the hand bent round so as to form a tube, or through the lips when disposed as if to whistle. The sound faintly partakes of a reverberating character. In some forms of disease the respiration acquires a much more intense resounding hollowness: a kind of bronchial

breathing to which Laennec gave the name Cavernous. Cavernous breathing is simulated when, having placed both hands over the mouth so as to form a cup-shaped cavity, we blow gently through a small chink left open opposite to the mouth: or again when, having moved the tongue far back, we breathe through the lips pouted as if to pronounce the vowel *oo.* Sometimes bronchial breathing is not unlike the sound produced by respiration carried on through the mouth shaped as if to sound the vowel *au.* Indeed there are few kinds of bronchial or cavernous breathing which cannot be imitated by the mouth : a fact which will hereafter afford a clue to the acoustics of the matter. The loudness of bronchial breathing is an unimportant property: the special quality is as well marked in respiratory sounds which are weak as in the loudest; nay, loudness of sound often proves an impediment to the detection of that quality which alone constitutes the criterion of bronchial breathing. The expiration is always audible, although usually less loud than the inspiration : and, because less loud, the expiration manifests the special quality more clearly than the noisier inspiration.

A kind of bronchial respiration, which has

been named Tubular, is characterised by a well-marked whiffing quality, such as may be remotely imitated by breathing through the mouth disposed so as to pronounce the sharp aspirated guttural (the *ch* in the German word Becher), or the vowel *ee*; or by blowing sharply through the canal of a stethoscope. Tubular breathing is most frequently heard over hepatised lung, and, when once heard, is not likely to be forgotten.

By the name of souffle or puff, Laennec designated a phænomenon quite analogous to pectoriloquy, and which is sometimes superadded to bronchial, cavernous, or tubular breathing. The air during inspiration seems to be drawn away from the ear of the observer, and during expiration to be puffed back again.

Physical Conditions of Respiratory Sounds.

The breathing-sounds, which have now been described, originate in different parts of the respiratory apparatus.

i. The transit of air, through the narrow glottis, into the laryngeal cavity below, or the pharyngeal space above, produces sounds inspiratory and expiratory.[1] These glottidean sounds

[1] Beau: Traité d'Auscultation, 1856, pp. 14 *et seqq.*

are carried along the respiratory passages just as the vocal sounds are carried. All that has been said concerning the conduction of vocal resonance applies mutatis mutandis to the respiratory sounds.

ii. The passage of the breath, through the narrow mouths of the air-sacs into the wider cavities beyond, causes an inspiratory sound. And, if indeed there be a membranous fold around the orifice of each air-sac,[1] it is easy to understand how the expiratory movement likewise should produce a sound.

iii. When disease obliterates the air-vesicles and the smallest air-tubes proceeding from a larger bronchium, and so converts it into a cavity with unbroken walls, it becomes capable of resonance. When a current of air passing across the mouth of such a tube produces a commotion there, a certain pulse of this commotion is taken up by the resounding cavity, and raised into a definite sound.[2] An effect concurrent

Chauveau and Bondet: Gaz. Hebdom., 1863, Nos. 39 and 52. The immediate cause of murmurs in general is considered under the head of cardiac murmurs.

[1] Waters: British and For. Med. Chir. Rev. Jan. 1865.

[2] Bloodgood: Amer. Journ. Med. Science, 1860. See also page 65.

with the movements of both inspiration and expiration.

iv. Air entering an accidental cavity formed within the lung produces an inspiratory sound.

Concerning the application of these data to the explanation of what is actually heard by the auscultation of respiring lungs. Vesicular breathing is due in greater part to the sound produced in the air-sacs, but a smaller part (especially the expiration) depends upon conduction of the laryngeal sound. Ordinary bronchial breathing is simply the glottidean sound carried down the air-tubes. In some forms of consolidation the bronchial breathing is reinforced by the addition of a new sound produced by resonance in the manner described in the third of the foregoing paragraphs. In cavities the original laryngeal sound is modified both by resonance and by the addition of a sound altogether new.

¶ II. RESPIRATORY SOUNDS IN HEALTH.

The breath-sound is vesicular over the whole of the pulmonary regions: excepting that bronchial breathing is to be heard between the scapulæ in many persons who are perfectly healthy; sometimes under the right clavicle

also (especially in women); and, still more rarely, under the left. In children, the respiratory sound is loud or puerile; a fact to be explained by the frequency and depth of their respiration, by the thinness of their chest walls, and by the perfection of the membranous septa of their lungs. The rumbling sound produced by involuntary fibrillary contraction of the muscles of the chest is often heard during auscultation, but being continuous cannot be confounded with the sounds of respiration. A crepitation due to collapse is sometimes heard, especially at the bases of the lungs, and at the commencement of an examination; a few deep breaths suffice for the permanent removal of this rale.

¶ III. RESPIRATORY SOUNDS IN DISEASE.

I. Weak respiration indicates diminution in the ebb and flow of air in the lungs. Whatever obstructs the air passages within, whatever compresses them without, whatever interferes with the movements of the chest, will produce weak respiration; which, in other respects, may be vesicular or may be bronchial. That form of weak breathing which has been called deferred

inspiration, occurs especially in emphysema and in laryngeal obstruction.

II. Loud respiration, when a consequence of disease, indicates that the breathing is obstructed in some part of the lungs other than that where the loud respiration is heard.

III. Bronchial respiration, heard where naturally the breathing is vesicular, indicates obliteration of the spongy structure of the lung: obliteration which may be brought about in two ways:—

i. By collapse of the air-sacs, or by exudation into them, or by both processes combined. The pulmonary substance, thus rendered more homogeneous, is better fitted to convey the sounds generated in the larger bronchia, to the surface. Pneumonic, phthisical, tubercular, hæmorrhagic, cancerous consolidations; collapse, simple, congestive, or œdematous; and thickening of the bronchial tubes with condensed surrounding tissue; all produce this effect.

ii. By destruction of the air-sacs: whereby the vesicular structure is replaced by large cavities capable of a high degree of resonance. Phthisis, and dilatation of the bronchi, are the most frequent lesions belonging to this class.

Wherefore, in brief, consolidations and cavities

are the chief causes of bronchial breathing, as also of bronchophony.

APPENDIX.

Rales.

The natural breath-sound having been described, and also the modifications of that sound which occur in disease, I now come to Rales or sounds which are produced within the lung by respiration, and which are wholly additional to the natural or modified breath-sound. Rale, rhonchus, rattle; these words have been used synonymously. I shall retain the classification adopted by Laennec, and also his nomenclature, because I believe that his distinctions are of practical value in diagnosis, and that his names are as good as any others.

Rales are of three kinds; crepitant, mucous, and sonorosibilant.

I.—*Crepitant Rale.*

A rale which is quite comparable to the sound produced by rubbing a lock of the hair between the fingers close to the ear. Crepitation of this kind is rarely heard except during inspiration,

sometimes throughout the whole of it, sometimes towards the end of it only. Pneumonia, collapse, and œdema of the lung, are the three conditions which afford a crepitant rale. Its occurrence in collapsed lung, during a deep-drawn breath, affords, I believe, the key to the immediate physical condition necessary to the production of crepitation in most cases; namely, the opening-up of collapsed air-sacs.[1] Disseminated collapse of single air-vesicles is an important item in the changes consequent upon pneumonia and pulmonary œdema, as any one who will inflate the uncut engorged or œdematous lung of a child may easily discover. Insufflation of the lung in such cases will bring out an immense number of air-sacs, which were before invisible because collapsed, and which collapse again directly the air is allowed to escape: the transparent, non-pigmented tissues of a child are particularly favourable for this experiment. In pneumonia as soon as the hepatisation becomes dense the collapse cannot be removed by any pressure of air. Sometimes,

[1] Van Swieten: Comm. in Aphor. § 826. "Ingratus in pectore strepitus, qui fit a vesiculis pulmonum siccis hincque crepitantibus instar corii arefacti, dum inspirando extenduntur."

doubtless, crepitation is a very fine mucous rale.

II.—*Mucous Rale.*

This rale includes all the sounds which are due to the passage of air through liquids contained in the air-passages. The notion received is certainly, for the most part, that of the bubbling of liquids, but sometimes the sound is crackling rather than bubbling in character. The mucous rale is subdivided into varieties, according to the following considerations:—

i. The apparent size of the bubbles: so small as to approach the crepitant rale (subcrepitant), so large as to deserve the name of gurgling, and all intermediate sizes.

ii. The distinctness of the rale: the rale being sometimes more or less obscure, on account of the weakness of the respiration; a deeper breath (when this is possible) bringing out a rale much more distinct.

iii. The resonance of the rale: which is sometimes so clear as quite to deserve the name of bronchophonic. Resonant mucous rales indicate the same physical conditions as do resonant (bronchial) breathing or voice.

III.—*Sonorous and Sibilant Rales.*

These are rales which are more or less accurately described by such words as snoring, cooing, whistling, hissing; the long low-pitched sounds being called sonorous (rhonchus), and the short high-pitched, sibilant (sibilus). Sounds of this kind are due to local narrowing of the air-passages; most commonly by mucus, in which case a cough which dislodges the mucus removes the rale. Palpable fremitus often concurs with these rales.

IV.—*Doubtful Rales.*

The respiratory sound is sometimes attended by sounds not comprehended in any species of rale hitherto described, and doubtful both as to situation and significance. I allude particularly to two kinds of sounds. First: sundry creaking sounds not rarely heard at the apices of the lungs, and possibly due to creaking of pleural adhesions, but possibly also produced in the tissue of the lung itself. Secondly: the Dry crepitant rale with great bubbles, as Laennec named a sound resembling that produced by inflating a dried bladder, and probably really

due, as he supposed, to distension of the enlarged air-sacs of emphysematous lung.

Article III.—Pleural Sounds.

The pleuræ in a state of health can generate no sound.

In diseases of the pleura sundry sounds are met with, which may be arranged in two classes; friction sounds, and sounds produced in large cavities.

¶ I.—FRICTION SOUNDS.

Any unevenness of opposed pleural surfaces tends to make a friction or rubbing sound accompany the respiratory movements.

The recognition of friction sounds is the most important addition to the practice of auscultation of the lungs, as it was left us by Laennec.[1] Not that he was altogether ignorant of these sounds. In the spring of 1824, M. Honoré, who had succeeded Laennec at the Hôpital Necker, having observed a peculiar sound in the chest of a patient, sent him for that reason to Laennec; who heard what he called "up and

[1] I have elsewhere alluded to the fact that Hippocrates was acquainted with pleural friction-sounds.

down rubbing," and considered it to be due to interlobular emphysema. The real meaning of pleural friction was first made out by Reynaud. The diagnosis of a friction sound depends upon a consideration of the following particulars:—

1st. The Character of the sound.

i. This is sometimes quite peculiar, and special to pleural friction; giving a distinct notion, either of rubbing in any grade of intensity, between the lightest grazing and the harshest scraping; or of creaking, comparable to the creaking of leather.

ii. Sometimes friction sounds are quite indistinguishable, per se, from the rales produced in the air-passages: extra-pulmonary adventitious sounds closely simulate the intra-pulmonary. Crepitant and sharp small mucous rales are the rales which are most frequently imitated by friction sounds. In such cases the diagnosis depends upon other circumstances than the mere character of the sound.

2nd. The Position of the sound.

A friction sound is commonly heard over a small part of one side of the chest; especially the lower half of the chest below the axilla,

[1] In 1829. I have not been able to procure the original paper in the Journal Hebdomadaire of that year.

or about the angle of the scapula; here the movements of the pleural surfaces upon each other are probably the freest. Sometimes a friction sound is heard over the whole of one side, and then mostly possesses the character of a false rale. It is rare to hear friction sound on both sides of the chest at the same time.

3rd. The Time of the sound.

A friction sound may accompany inspiration, or expiration, or both, or only a small portion of either, especially the very end of inspiration. So that friction sound is mostly of respiratory rhythm; but occasionally pleural friction may be developed over the præcordial region by the action of the heart, in which case the rhythm is cardiac.

4th. Friction sound is mostly an isolated phænomenon, that is, is not accompanied by any unnatural quality of respiratory or vocal sound.

5th. Cough has no power of modifying or removing friction sounds.

A person acquainted with these facts will mostly be able to make a satisfactory diagnosis; especially if he take the course and symptoms also, of any given case, into consideration. The

commonest cause of roughness of the pleura is exuded lymph; miliary tubercles, and mere extra-vascularity, may likewise cause a friction sound.

A friction sound is not necessarily abolished by the co-existence of a very considerable amount of serous effusion. Laennec believed (as has been said) that a friction sound was producible by interlobular emphysema, and probably he was not mistaken. Dr. Walshe is of the same opinion,[1] and Dr. W. T. Gairdner, in a case of vesicular emphysema without adhesions, heard a friction sound of "shuffling" character, and felt a distinct rubbing.[2]

Sound of Pulmonary Shock. Aran [3] has described, under this name, an excessively harsh friction sound, attended by palpable fremitus, produced by cough, and due to the collision of lung and thoracic wall in certain cases of pleurisy complicated with pneumothorax.

[1] Diseases of the Lungs. 3rd edit. 1860, page 133.
[2] Clinical Medicine, 1862, p. 438.
[3] Arch. Gén. de Méd. 1856.

K 2

¶ II.—AMPHORIC SOUNDS:

Or, sounds produced in a large cavity, are four in number: two spontaneous, and two produced by the physician at the moment of examination: the former including amphoric echo, and metallic tinkling; the latter including the bell sound, and the succussion splash. I describe these signs in this place because they are most frequently met with in the pleural cavity.

I.—AMPHORIC ECHO.

By Amphoric echo is meant an intense metallic resonance, such as is possessed by the sound produced by blowing, speaking, or coughing, into a large and empty glass bottle. An amphoric echo requires the existence of a large cavity containing air; and may accompany—

i. The respiratory sounds; when there is a wide fistulous opening between a pneumothorax and a large bronchus, so that the breath passes freely in and out; or when loud respiratory sounds are merely generated close to a large resounding cavity, they acquire an amphoric quality.

ii. The voice, cough, and rales.

iii. The cardiac sounds heard through a pneumothorax.

iv. The sound produced by swallowing: the amphoric echo attending this sound will be heard on auscultation of a stomach, greatly distended by air, while the patient is swallowing a few drops of liquid.

II.—METALLIC TINKLING.

A sound compared by Laennec to that which is produced in a metallic, glass, or porcelain cup when struck gently by a pin. Metallic tinkling is a single sound, which attends respiration, speaking, coughing, or even mere change in the position of the body. With respiration the sound is mostly intermittent, that is, not heard with each movement; with speaking, the sound is produced more constantly, especially if the patient speak slowly, with a period between each syllable; with coughing, the sound is tolerably constant. The sound is heard on one side only, and mostly heard best in the middle zone of the side; sometimes it is heard best at the apex, and is then of limited extent.

III.—BELL SOUND.

This sign, although not unknown to Laennec,

was first more particularly pointed out by Trousseau.[1] It is heard by auscultation of a large air-containing cavity, whilst an assistant strikes the surface of the sac by means of a metallic pleximeter and hammer, for which purpose two coins answer perfectly well. Under these conditions a beautifully clear ringing sound will be heard, in tone not much inferior to that of the bell of a small clock.

IV.—SPLASHING SOUND.

This most venerable physical sign was well known to Hippocrates.[2] When a large cavity contains both liquid and air, and the patient is shaken while the physician applies his ear to the chest, a splashing sound is heard. In fact the splash is often heard by the patient himself.

PHYSICAL CONDITIONS OF AMPHORIC SOUNDS.

I have now spoken of the last of a most interesting series of signs afforded by large resounding cavities. I have classed these signs under the head of pleural sounds, because it is in the pleura that the physical conditions necessary

[1] Gaz. des Hôpitaux, 1857, page 157, "Bruit d'airain."
[2] De Morbis: passim. See page 98, note.

to their existence are most frequently met with. These necessary physical conditions are the following:—

i. *A large Cavity.*—For the production of the bell sound the cavity must be very large; at least, in pneumothorax artificially produced in the pleuræ of children post mortem, I have not heard the sound; nor in distension of the stomach, unless extreme; nor in hollow caoutchouc balls of four inches diameter. The metallic tinkle may be heard, according to Kolisko,[1] and Wintrich, in a cavity not larger than a pigeon's egg, provided that the cavity be connected with a large air-tube. The amphoric echo and the Hippocratic splash may occur in cavities of moderate size. The walls of the cavity must be smooth.

ii. *The Presence of Air in the Cavity.*—The succussion splash obviously demands the presence of liquid also. For the amphoric echo and bell sound, air alone suffices; for the latter indeed the quantity of air required is so large that the presence of much liquid in the cavity would counteract the sign. Laennec supposed that metallic tinkling did not occur except both

[1] Skoda: Abhandlung, etc. Markham's translation, p. 135.

air and liquid were present in the cavity; Skoda denies the necessity of liquid; the question is hardly worth debate: the intensest amphoric echo of respiratory sounds, or of rales; the falling of a drop of liquid; the bursting of a bubble; and crackling friction-rale; all are possible causes of the metallic tinkle.

iii. A communication between the bronchi and the cavity is certainly not essential to the production of any of the sounds.

From the foregoing principles it will be now clear, why the amphoric echo and metallic tinkle occur in large phthisical cavities, and in pneumothorax: why the bell sound is not heard except in pneumothorax: and why the splash may be produced in hydropneumothorax, in large phthisical, suppurative, or gangrenous cavities, and in hydropneumopericardium. The distended stomach sometimes affords the means of demonstrating all of the amphoric sounds: the colon will occasionally give a succussion splash and the bell sound.

SECTION III.

AUSCULTATION OF THE HEART.

Laennec brought the auscultation of the lungs well nigh to perfection, but it was far otherwise with the auscultation of the heart. He confirmed the truth of Harvey's remark, that sound is produced by the action of the heart:[1] he noted that this sound is double: he discovered the existence of murmurs, or unnatural sounds: but when he attempted to go further, and to explain the sounds and the murmurs, he became lost in confusion; 'seeking a way and straying from the way; not knowing how to find the open air but toiling desperately to find it out.' He even went backward; specially by denying, in the second edition of his work, what he had maintained in the first, that at least some thrills and some murmurs are associated with valvular obstruction.

A succession of physicians since Laennec's day have contributed to bring the auscultation of the heart into its present satisfactory state. The gradual correction of Laennec's fundamental error concerning the time of the auricular sys-

[1] De motu cordis, cap. v.

tole: the origination of the true theory of the heart-sounds by Sir Robert Carswell, and its extension by Dr. Billing and Rouanet:[1] the discovery of pericardial friction by Collin,[2] and the development of this subject by Dr. Stokes:[3] the discovery of aortic regurgitation by Sir Dominic Corrigan:[4] the explanation of the significance of præsystolic murmurs by Fauvel:[5] the establishment of the doctrine of the localisation of murmurs; and lastly the invention of the sphygmograph and cardiograph by Marey and Chauveau:[6] these have been the most important steps in the progress of our knowledge.

The movements of the heart are attended by certain sounds which, according as they are produced within the heart or outside it, are named endocardial or pericardial.

[1] See page 140.

[2] Collin: Des diverses méthodes d'exploration de la poitrine. Paris, 1824. Quoted by Stokes: Diseases of Heart, 1854, page 8.

[3] Stokes: Researches on the diagnosis of pericarditis. Dublin Journ. Med. Science, 1833.

[4] Corrigan: Edinb. Med. and Surg. Journal, 1832.

[5] Fauvel: Rétrécissement de l'orifice auriculo-ventriculaire gauche. Arch. de Méd. 1843.

[6] See page 48.

Article I.—Endocardial Sounds.

¶ I. ENDOCARDIAL SOUNDS IN HEALTH.

The ear applied to the præcordia of a healthy person will perceive that, for each impulse of the heart felt, two sounds are heard. One sound accompanies the impulse, and one follows it: wherefore the former is called the first or systolic sound; the latter, the second or diastolic. The two sounds differ in character; the second being sharper and shorter than the first. The first sound is heard loudest over the fourth or fifth intercostal spaces just within the left nipple line: the second, at the base of the heart, opposite the third costal cartilages.[1]

[1] In reality the heart-sounds are four in number, two systolic (mitral and tricuspid), and two diastolic (aortic and pulmonary). Skoda has always taught that the sounds of the heart are eight, two (systolic and diastolic) produced at each orifice. The most recent German writers reduce the number to six, but do not agree concerning the two which are additional to the four above spoken of. Von Dusch and Bamberger maintain the existence of aortic and pulmonary systolic sounds: Gerhardt, on the other hand, of mitral and tricuspid diastolic.

Causes of the Heart's Sounds.

I believe that, at present, we must accept that doctrine which refers the sounds of the heart to sudden vibratile tension of the valvular flaps. The first sound is caused by vibration of the closed auriculo-ventricular or cuspid valves, and is of a duration almost equal to that of the ventricular systole. The second sound is caused by closure of the ventriculo-arterial or sigmoid valves, and attends the beginning of the ventricular diastole. This theory of the second sound was first announced by Sir Robert Carswell in 1831 :[1] and, the next year, Dr. Billing,[2] and Rouanet,[3] each independently of the other,

[1] Marc d'Espine: Recherches sur le diagnostic des maladies du cœur. Arch. Gén. de Méd. Oct. 1831.

[2] Paper read at Anniversary Meeting of the Hunterian Society, 1832. Med. chir. Review, April 1, 1833. "The push of the heart is caused by the swelling up of the ventricular muscles in their systole to expel the blood; the first sound is caused by the tension produced in shutting the auriculo-ventricular valves, and the second sound is caused by the tension produced in the shutting of the arterio-ventricular valves."

[3] Analyse des bruits du cœur. Aug. 1832. Thèse inaugurale. See: Notice scientifique sur Rouanet de Saint-Pons (Hérault): par le Dr. Faget. L'Union Méd. 1866.

extended the same explanation to the first sound.[1]

Loudness of the Sounds.

i. Whatever weakens the heart weakens the sounds. And weakens the first sound especially, inasmuch as it is more immediately connected with the action of the heart itself: whereas the second sound is due to the elastic recoil of the large arteries. In continued fevers, for instance, the first sound sometimes becomes inaudible.

ii. But the loudness of the sounds, as we hear them, depends not only upon the state of the heart itself, but also upon the quantity of material interposed between the heart and the ear; wherefore in very fat persons, in emphysema and in pericardial effusion, the sounds are weak.

[1] I have adopted this explanation because, while being at least as plausible as any other, it is peculiarly adapted to clinical teaching. The cause of the first sound seems to be the unconquerable Caledonia of physiology: quite recently Ludwig has revived Laennec's muscular theory: fortunately the subject has not much bearing upon practical medicine.

Reduplication of the Sounds.

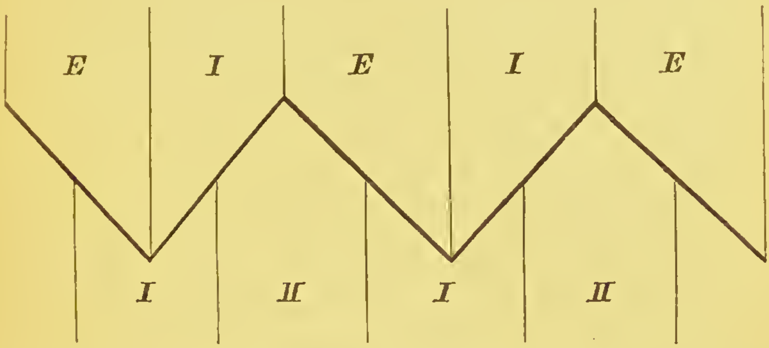
A heart-sound is said to be reduplicate when, instead of being a single sound, it is more or less doubled or repeated. Occasionally the sound is completely doubled, that is to say, in the place of one sound two sounds are heard, with a distinct interval between them: but much more frequently the reduplication is incomplete, that is to say, before the first portion of the doubled sound is concluded the second begins. Thus, if the single heart sound be represented by the syllable tup; the reduplicated sound would answer to tup-tup (complete), or turrup (incomplete).

The theory of reduplication has been elaborately expounded by Potain.[1] Reduplication of the sounds is very common at all ages, and is as common with one sound as the other. Intermittence is an almost constant character of reduplication, the sound being doubled with some beats of the heart and not with others. This intermittence is found to have a close relation with the movements of respiration: the first

[1] Note sur les dédoublements normaux des bruits du cœur. L'Union Méd. 1866.

sound doubles at the end of expiration and the beginning of inspiration: the second at the end

Fig. 12.



REDUPLICATION OF HEART'S SOUNDS.

E = expiration. *I* = inspiration. The figures refer to the sounds of the heart.

of inspiration and the beginning of expiration.[1] The reduplication is due to asynchronism in the closure of homologous valves; the two cuspid valves, or the two sigmoid valves, as the case may be, are not shut at precisely the same time: the asynchronism is due to difference in the amount of pressure exerted upon the valves: and the differences of pressure depend upon the different states of respiration. I will not follow M. Potain into the further

[1] A fact previously noted by Schafer: Canst. Jhb. 1860, ii. 99.

development of his theory, which becomes somewhat subtile, and is, I think, open to adverse criticism. I will only add that, in laborious respiration, the order of the reduplications is exactly reversed: the first sound being doubled at the end of inspiration and beginning of expiration; and the second sound at the end of expiration and beginning of inspiration.

The practical value of these considerations is this: that, by observing whether apparent reduplications follow the law above stated or not, we can distinguish true doubling of the sounds from those false reduplications which are in reality compounded of a sound and a murmur.

¶ II. ENDOCARDIAL SOUNDS IN DISEASE.

Any fundamental change in the character of a heart-sound, or any superadded sound, constitutes a murmur, or unnatural endocardial sound: pericardial sounds excepted. The diagnosis between the unnatural sounds produced within the heart, and those produced outside it, will be considered under the head of pericardial sounds.

The immediate cause of murmurs is usually to be found in some alteration of the relationship which naturally exists between the cardiac

orifices and the blood. The orifices both admit the blood and shut it off; and both when open and when closed may give rise to murmurs. An orifice which is open gives rise to a murmur when it affords any obstruction to the free passage of the blood: and an orifice which should naturally be closed gives rise to a murmur when it is not closed, but allows the blood to pass back into the cavity whence it came. Murmurs produced at an open orifice, and in the natural current of the blood, are called onward or obstructive murmurs: murmurs produced at a closed orifice, and against the natural current of the blood, are called backward or regurgitant murmurs. Murmurs of the first kind are new sounds: murmurs of the second kind are merely altered sounds.

The physical condition of murmurs is generally to be found in the abrupt passage of fluid from a narrow into a wider space; the murmur is produced just beyond the narrow spot. This explanation, which we owe to Corrigan,[1] is applicable to many murmurs produced in the air-passages, as well as to cardiac and vascular murmurs.

[1] Inquiry into the causes of the Bruit de soufflet and Frémissement cataire. Lancet; April 4, 1829.

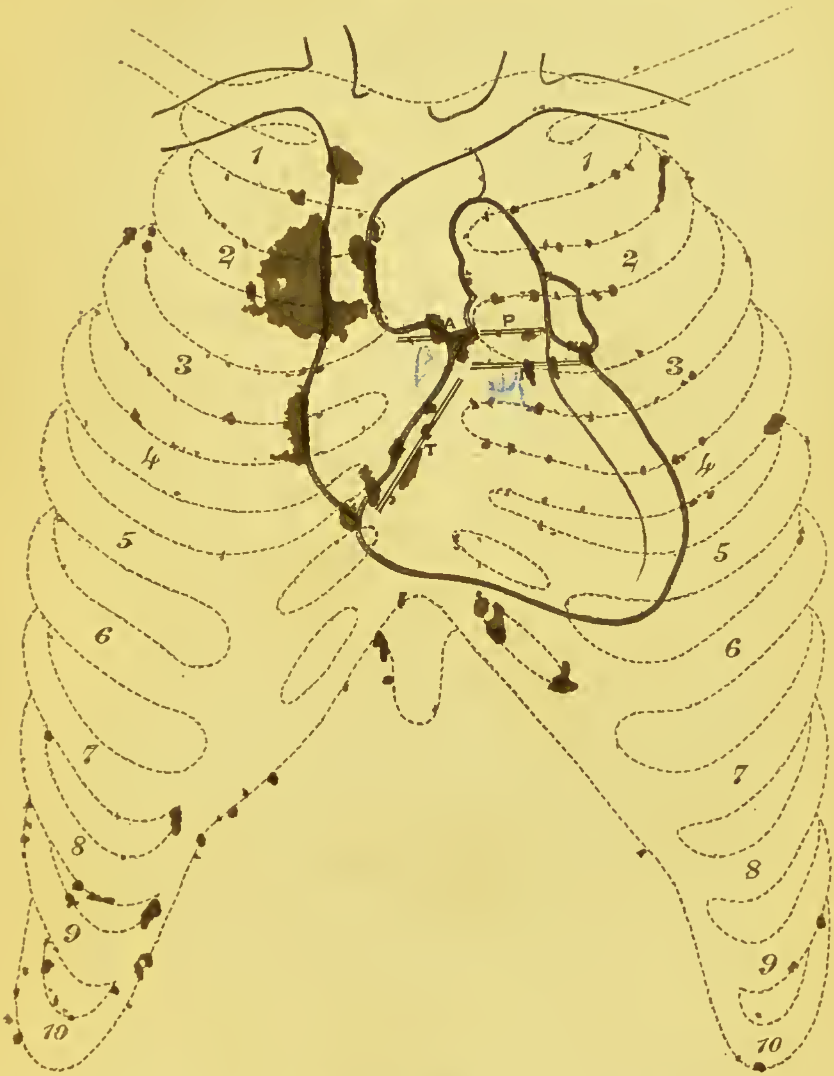
So far I have spoken of murmurs which own an obvious physical cause; organic murmurs: but many murmurs there are which cannot be explained in this way, nor indeed at present explained at all; these murmurs are called inorganic, and will be spoken of hereafter under that name.

Murmurs were once characterised according to their acoustic qualities, whether blowing, filing, rasping, sawing; but these are vain distinctions: in order to render murmurs serviceable in the diagnosis of disease we now regard only two things, their Place and Time.

1st.—The Place of Murmurs.

Generally speaking, a murmur is heard best at that point of the surface of the body which is nearest to the orifice whereat the murmur is generated. So that it becomes highly important to study the relation which the orifices of the heart bear to the chest wall. The annexed diagram illustrates these facts. The Pulmonary orifice lies behind the second left interspace, close to the sternum: the Aortic orifice lies on the same level, behind the sternum: the Tricuspid orifice reaches from the sternal end of the third left rib to that of the fifth right rib: the

Fig. 13.



POSITION OF HEART WITH REFERENCE TO CHEST-WALLS.

A = site of aortic valve. P = pulmonary.
M = mitral. T = tricuspid.

The direction of the rings, to which the bases of the aortic and mitral segments are attached, is obliquely upwards from right to left.

Mitral orifice lies on a level with the upper border of the third left cartilage. And let it be noted that the mitral orifice lies much more deeply than the rest.[1]

But murmurs are not always conducted the shortest way to the surface. The conducting power of the tissues interposed between a valve and the chest wall, and the direction of the blood current, have much to do with determining the point at which a murmur is heard loudest. i. The influence of the superjacent tissues is well exemplified in the case of aortic and mitral murmurs. Both orifices of the left side of the heart lie deep in the chest. The aorta becomes most superficial just above its valves, and is there almost in contact with a good conductor of sound, the sternum: wherefore aortic murmurs are well conducted up and down the sternum, and along the attached cartilages: so that, in fact, an aortic murmur is sometimes heard louder at the xiphoid cartilage than at the second intercostal space. The mitral orifice, buried beneath heart and lung

[1] See the plates of Luschka or Sibson. Or Von Dusch: Lehrbuch der Herzkrankheiten. Leipzig, 1868, for a very minute description of the relation which the sundry parts of the heart bear to the anterior chest wall.

tissues, may be said to become practically most superficial where the cavity of the left ventricle becomes most superficial, that is to say, at or just above the apex-beat of the heart; and here, as a rule, mitral murmurs are heard loudest. ii. The influence of the blood-current (or of convection, as Dr. Salter has proposed to call it[1]) is exemplified best in the case of aortic murmurs. Both obstructive and regurgitant aortic murmurs are well conveyed by the blood along the arteries, and regurgitating murmurs may be conveyed by the reflux of blood towards the apex of the left ventricle.

2ndly.—The Time of Murmurs.

A complete cycle of the heart's action may be divided into four periods. i. The Auricular systole: immediately preceding the impulse, and not attended in health by any sound: the auricles contracting. ii. The Ventricular systole: accompanied by the impulse and the first sound: the ventricles contracting. iii. The Arterial systole: following the impulse, and attended by the second sound: the aorta and

[1] On cases illustrating the influence of convection in determining the seat of cardiac murmurs. Lancet, Aug. 1869.

pulmonary artery contracting. iv. The period during which all parts of the heart are at rest. The subject requires a further development, thus :—

		Auricular Systole.	Ventricular Systole.	Arterial Systole.	Rest.
Relative duration. Pulse=60.		$\frac{1}{10}$	$\frac{4}{10}$	$\frac{5}{10}$	
			impulse.		
Valves	cuspid	open	shut.		
	sigmoid		open	shut.	
Sounds			first	second.	
Murmurs		præsystolic	systolic	diastolic.	

3rdly.—Diagnostic Signification of Organic Murmurs.

i. Systolic murmurs referable (α) to the Auriculo-Ventricular orifices indicate reflux of blood : (β) to the Arterial orifices indicate constriction at or just above the orifice, unevenness of the conus arteriosus, dilatation of the aorta, perforation of the septum ventriculorum, or dilatation of the ductus arteriosus.

ii. Diastolic (and præsystolic) murmurs produced at the Cuspid orifices indicate constriction or obstruction of the orifice : at the Sigmoid

orifices indicate reflux of blood, or dilatation of the vessel above the valve.

The pathologic conditions leading to organic endocardial murmurs are the following: endo-
carditis acute or chronic (including the athe- 1
romatous process); thrombi, vegetations, or 2 3
tumours in the cavities or orifices; primary 4
dilatation of the heart or aorta; laceration of a 5
valvular segment; rupture of chordæ tendineæ, 6
leading to reversibility of cuspid flaps; atrophy 7
of the sigmoid valves, leading to their reversi-
bility; atheroma of the sinuses of Valsalva, 8
whereby the sigmoids are not evenly adapted
to each other, so that one or two segments have
to bear the whole pressure of the blood and are,
by it, retroverted;[1] and congenital malforma- 9
tions.

Murmurs, audible in the horizontal posture, sometimes become much less loud in the erect, nay, occasionally altogether cease. This is most frequently the case with murmurs produced at the mitral orifice. The reason of the occurrence is not quite clear.

Pressure upon the præcordia sometimes makes a murmur weaker. On the other hand, as has

[1] Gull: Guy's Hosp. Reports, 1859.

been shown by Latham[1] and Jenner,[2] pressure over the pulmonary artery will actually beget a systolic murmur in young persons.

Article II.—Pericardial Sounds.

No sound is produced by the movement of healthy pericardial surfaces upon each other.

In diseased states of the pericardium two kinds of sounds may be heard: friction sounds, and sounds due to the presence of air and liquid in the cavity.

¶ I.—PERICARDIAL FRICTION.

It is convenient to consider pericardial friction sounds with especial reference to their diagnosis from endocardial sounds. The most important diagnostic characters of friction sounds are these:—

i. The special quality sometimes suffices for the diagnosis, being distinctly rubbing or scraping: but often enough this special quality is ill-marked or absent. ii. Friction sounds are mostly of limited extent, heard over a small portion only of the præcordia, especially at the

[1] Diseases of Heart. 1846, vol. i. p. 62.

[2] On the influence of Pressure, &c. Med. Times and Gaz., Mar. 1856.

base of the heart; they do not follow the laws laid down with regard to the points of greatest intensity, and the conduction of endocardial murmurs. But a very loud friction sound may be heard, not only all over the præcordia, but even all over the front of the chest, nay even at the angle of the scapula: though, in these latter cases, there is mostly marked and sudden weakening of the sound as soon as we pass away from its place of origin. iii. Friction sound is mostly both systolic and diastolic, being loudest in the systole; sometimes systolic only, or even diastolic only. A sound, which is at one time systolic and anon diastolic, is pericardial. A sound, which has no definite relationship with either of the heart-sounds, is probably pericardial. iv. Friction sounds are not intermittent like reduplications. v. The unchanged heart-sounds are sometimes heard through friction. vi. Friction sounds often give a notion of superficiality, be it explained as it may. vii. Palpable vibration sometimes attends them. viii. Their loudness is increased by deep inspiration, which often weakens an endocardial sound. ix. Their loudness is sometimes increased by pressure, may however be diminished: moreover pressure sometimes influences the loudness

of endocardial murmurs. x. Friction sounds may be modified by change in the position of the body. Their place of greatest loudness may be thus made to change: this is very characteristic.[1] Friction sound sometimes disappears when the patient sits up, possibly on account of a small quantity of fluid which comes forward so as to separate the pericardial surfaces: but it has been shown that the same change in position sometimes removes endocardial murmurs. A friction sound, not to be heard otherwise, is sometimes produced when the patient lies upon his left side, whereby the position of the heart is much changed. A sound which shifts in place, from day to day, is pericardial.

Whatever roughens the pericardium can produce a friction sound: mere excessive vascularity, exudations, hæmorrhages, adhesions.

¶ II. Splashing sound has been heard in hydro-pneumopericardium.

[1] Oppolzer: Vorlesungen, i. 26.

APPENDIX.

Sounds, neither Endocardial nor Pericardial, produced by the Action of the Heart.

i. Respiratory sounds, due to the action of the heart upon the lung, are occasionally heard in the neighbourhood of the præcordia. They sometimes possess the character of vesicular breathing, but are sometimes blowing, whiffing, crackling, or rasping. They are mostly systolic and inspiratory; that is, the inspiratory sound is rendered more loud during each systole. They usually cease to be produced when the breath is held. A pulmonary cavity, near enough to the heart to be influenced by it, sometimes affords a loud systolic murmur,[1] and the same result has been observed in pneumothorax.

ii. Rales, in rare cases, have been produced by the movements of the heart upon the air-tubes.

iii. Friction sound generated in the pleura, of both respiratory and cardiac rhythm, sometimes is heard. Friction heard behind or alongside

[1] But see page 179.

the sternum, from the second to the sixth rib, is always pericardial. When a friction sound is heard at other parts of the præcordia, the diagnosis is not easy: pleural friction usually ceases when the breath is held, but this is not always the case; and there is good reason for believing that true pericardial friction may be occasionally arrested in that manner.

Auscultation as applied to the large vessels of the mediastina will be treated of hereafter.

CHAPTER VI.

APPENDIX.

SECTION I.

AUSCULTATION OF THE ARTERIES.

THE sounds of the heart, especially the second sound, are conducted along the arteries; not usually, however, farther than the carotids

and subclavians. Yet, as hereafter mentioned, very shrill diastolic murmurs produced at the aortic orifice, may sometimes be heard in the radials.

True Arterial Sounds : Sounds which are produced in the arteries themselves at the spot ausculted.

i. Pressure murmurs.—In healthy persons, a slight amount of pressure by the stethoscope upon the larger arteries will generate a soft tone, systolic with reference to the left ventricle. In some forms of disease, notably in hypertrophy of the left ventricle and in chlorosis, firmer pressure converts the soft tone into a harsh whizzing murmur. Moreover, under those circumstances, a murmur is producible in the smaller arteries; for example, in the plantar and the volar.

ii. Subclavian murmurs.—A systolic murmur, seated in the subclavian artery, is frequently to be heard below the clavicles, especially on the left side. This murmur is perfectly similar to that which is produced in other arteries by pressure, and is therefore presumed to be due to the same cause. But the exact anatomical condition which brings the sound about is unknown; adhesions at the apex of the lung have

been suggested ; certainly the murmur is more commonly met with in persons who are phthisical or predisposed to phthisis than in others.

The physical cause of arterial murmurs is the same as that of murmurs in general, namely, passage of the blood from a narrowed to a wider space : the loudness of the sound is proportionate to the strength and swiftness of the current ; a law which will probably explain the fact that carotid murmurs are sometimes audible during expiration only, that is when the arterial stream is most powerful.

iii. Duroziez[1] has shown that, in some diseases, compression of the large arteries produces, not only a loud systolic murmur, but also a softer diastolic sound. The diastolic murmur is coincident with a certain degree only of pressure upon the artery ; which degree must be ascertained in each case by varying the amount of force used to compress the vessel. Duroziez' sign is best discovered in the femoral arteries : I have heard it in the axillary and brachial arteries. The causes of the double arterial sound are not exactly known. It is most constantly producible in aortic regurgitations : but

[1] Du double souffle intermittent crural, comme signe de l'insuffisance aortique. Arch. Gén. de Méd. 1861.

Duroziez himself now confesses[1] that it is sometimes present when there is no other reason to suspect valvular disease; this is especially the case in chronic poisoning with lead.

SECTION II.

INSPECTION OF THE VEINS OF THE NECK.

That the jugular veins sometimes afford valuable evidence of the existence of dilatation of the heart was known to Lancisi.[2] Of late years the whole subject of the physical signs to be seen and heard in the large veins has been submitted to very careful study.

In a healthy person, the internal jugular vein is not visible; the external jugular, on the con-

[1] Des maladies organiques du cœur et de l'aorte et du double souffle crural, d'origine saturnine. Gaz. des Hôpitaux. 1867, Nos. 146, 149, 150.

[2] Joh. M. Lancisii, De motu cordis et aneurysmatibus. Lugd. Bat. 1740. Propos. lvii. "Inquirere mechanicam rationem, ob quam in dilatationibus radicis Cavæ, Auriculæ, et Ventriculi dextri, ipsæ venæ Jugulares vicissim dilatentur, fluctuent, mirisque modis agitentur, et concidant." The cause is asserted to be a regurgitation of blood through the tricuspid valve. Lancisi refers to Hombert as having made the same observation in a paper published in the Proceedings of the Parisian Academy of Sciences in 1704.

trary, is visible, if not in the erect position, at any rate, in the recumbent. The external jugular vein usually possesses two sets of valves, one at its mouth, and one in the middle of its course. The internal jugular is provided with valves at, or a little above, its mouth. All these valves are very variable both as to number and position : moreover they are often quite incompetent to close the vessel; this is especially the case with the valve in the internal jugular. The right internal jugular vein, right innominate vein, and vena cava superior, form a continuous channel which is almost straight. For this reason, all the signs about to be described are more marked on the right side of the neck, than on the left; and they would be always more marked in the internal than in the external jugular, were it not that the deep position of the former vein is a hindrance to inspection.

Inspection of the veins of the neck is directed to two points; the fulness of the veins, and the movements which the contained blood undergoes.

¶ I. THE FULNESS OF THE VEINS.

I have already said that, in health, the external jugular alone is visible, and even that vein, oftentimes, only in the horizontal position of the body. In disease, both veins are sometimes dilated to the size of a finger. I shall call an unnatural fulness of the veins overfilling or distension of them.

Overfilling of the veins is either transitory or permanent.

i. Transitory overfilling accompanies powerful expiratory movements; which produce such an amount of pressure upon the intrathoracic veins, that the valves at the mouths of the jugular veins are shut, and the blood flowing down from above cannot pass into the innominata. Inspiration reverses all this; the veins are emptied, and collapse. Excess of expiratory muscular action, due to expiratory dyspnœa[1] or to cough, is the cause of the increased intrathoracic pressure. Repeated transitory overfilling of the veins is ultimately followed by permanent dilatation of them: a fact exem-

[1] See page 36.

plified by patients who suffer from chronic pulmonary catarrh. If, in these persons, when the cough is quiet and the veins are invisible, we place our finger just above the clavicle so as to obstruct the external jugular vein, it at once swells up and manifests the amount of its dilatation ; which may be taken as an index of the degree to which the patient's tissues have suffered in consequence of his cough.

ii. Permanent overfilling of the jugulars is mostly due to overfilling of the right auricle ; but, obviously, any obstruction to the upper vena cava, or to the innominata (by compression, thrombosis, or stricture) will have the same effect. When the original cause does not lie in the innominata, respiration affects the veins in the way above described, that is to say, they become more full during expiration, less full during inspiration. Permanent overfilling of the veins gradually causes them to dilate ; the valves become quite incompetent.

¶ II. MOVEMENTS WITHIN THE VEINS.

Besides the respiratory movements which have just been described, the blood contained within the veins frequently undergoes movements which are associated with the contractions of the heart. Venous pulsations are præsystolic or systolic.

i. Præsystolic pulsation in the veins is due to the auricular systole. Contraction of the auricle always sends a small quantity of blood back into the vena cava; but, naturally, the intrathoracic veins are not so full but that they can accommodate the additional quantity. When, however, these veins are quite full, any reflux of blood into them from the heart makes itself felt as high as the jugulars. Parrot declares that in a healthy person, when lying down, præsystolic pulsation can be seen in the jugulars. Be this as it may, when the veins are overfilled præsystolic pulsation is nearly always present, a fact proved by the sphygmograph. Whether the valves are competent or not makes no difference: when they are competent the impact of blood upon them from below is sufficient to agitate the blood contained in the veins above.

ii. Systolic pulsation in the veins is due to the ventricular systole, indirectly or directly. A direct systolic pulsation (venous pulse) signifies a reflux of blood out of the ventricle; the tricuspid valve being incompetent. When the valve is competent, we may call the pulsation indirect.

a. Indirect systolic pulsation is mostly due to the fact that the tricuspid valve is raised, by the ventricular systole, into a sort of dome, convex towards the auricle.[1] Hence an impulse backwards to the blood in the venous system; counteracted however, unless the overfilling of the veins is very great, by the diastole of the auricle. Friedreich suggests that in some cases the systolic filling of the aorta compresses the distended intrathoracic veins, and thus produces a movement in the jugulars. The pulsations of the carotid artery often enough communicate systolic movements to the veins; but compression of the vein at the clavicle does not stop movements of this kind, and does stop movements transmitted upwards along the vein.

β. Direct systolic pulsation, being due to the propulsion of a wave of blood from the left

[1] Marey and Chauveau have felt that this is really the case.

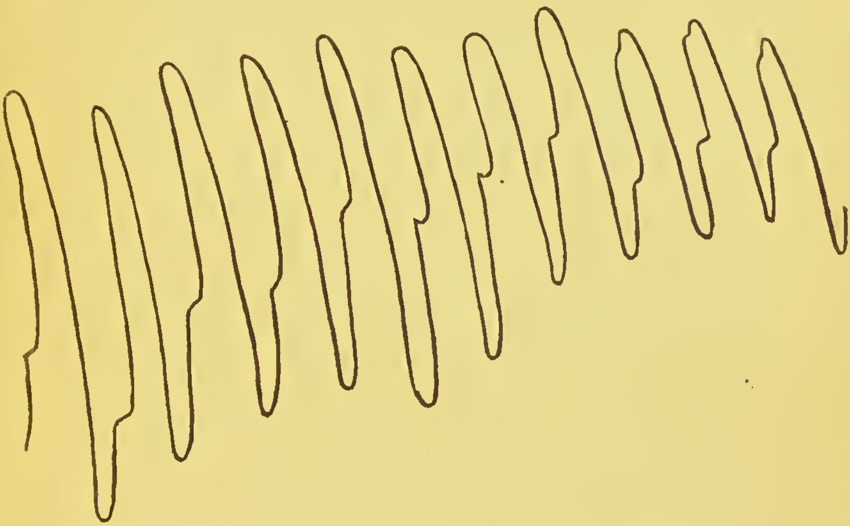
ventricle into the jugular veins, requires that both tricuspid and venous valves be incompetent. It is easy to ascertain whether the venous valves are competent or not; namely, by compressing the veins in the upper part of the neck, and observing whether they are filled with blood from below or not. But it is not so easy to determine reflux through the tricuspid valve; that is, to distinguish positively between direct and indirect systolic pulsation. The difference is only one of degree. Both pulsation and reflux are less marked when indirect than when the tricuspid valve is incompetent. In the latter case blood is pumped directly out of the ventricle into the veins under strong pressure. To repeat: when the pulse is strong there is probably tricuspid regurgitation; when the pulse is weak it may be independent of actual reflux from the right ventricle. When the pulse is very strong, it is sometimes palpable, or even thrilling.[1]

By Bamberger, Friedreich, and others, the sphygmograph has been applied to pulsating

[1] Cossy has described a case of anastomotic aneurysm, due to rupture of an aortic aneurysm into the vena cava superior: systolic pulse and thrill were present in the veins of the neck.

veins. The venous pulse has thereby been found to be dicrotous; but dicrotous in a

Fig. 14.



Tracing of Jugular Pulsation: from Friedreich.

manner different from the arterial pulse. The venous dicrotism is systolic, that is to say, it occurs in the rise of the blood wave. The first or smaller impulse coincides with the auricular contraction; the second or chief impulse with the systole of the ventricles. Sometimes there is a diastolic dicrotism at the very end of the fall of the pulse-wave: supposed to indicate repletion of the cavities of the right side of the

heart, a sudden stop being put to the entry of more blood.

Fig. 15.

TRACING OF JUGULAR PULSATION: SHOWING DIASTOLIC DICROTISM.

iii. Suddėn collapse of the jugular veins, during the ventricular diastole, has been shown by Friedreich [1] to occur in some cases of pericardial adhesion. The sign, which is always preceded by systolic recession of the chest walls (page 42), consists in this, that the veins, full during the systole, suddenly collapse so as to become almost or quite invisible during the diastole. The supraclavicular regions sometimes recede at the same time. Friedreich supposes

[1] Arch. für path. Anat. 1864. Also Die Krankheiten des Herzens. Erlangen, 1867.

that the diaphragm, drawn upwards by the adherent heart during the systole, and returning to its former position during the diastole, elongates the intrathoracic veins, and so sucks the blood out of the jugulars.

SECTION III.

AUSCULTATION OF THE VEINS OF THE NECK.

By means of a stethoscope, placed upon the side of the neck, there is often to be heard a humming sound; which was first referred to the veins by Dr. Ogier Ward.[1] This venous hum is usually continuous; but other murmurs, which are intermittent, are occasionally heard in the veins.

¶ I. The Continuous Venous Hum, although especially well heard in chlorotic patients, occurs in a large number of persons who are perfectly healthy. The pressure of the stethoscope is doubtless the immediate cause of the murmur. The more rapid the flow of blood the louder the hum: hence it is louder in the erect than in the lying posture; and is arrested by whatever produces stagnation of blood in the vein. Hence

[1] On the Bruit du diable. Lond. Med. Gaz. 1837, page 7.

also, in most cases, although the murmur is continuous, yet it is subject to rhythmical intensifications of loudness; which occur both during the inspiratory draught of blood from out the veins; and during the ventricular systole, that is, during the commencing auricular diastole, when the venous current begins again to flow freely. In anæmia, the relaxation of the capillaries causes the blood-currents to be unusually swift.

¶ II. Intermitting Venous Murmurs are præsystolic, systolic, or diastolic. They all are heard better on the right side than on the left.

i. Præsystolic murmurs due to the passage of blood backwards through the mouth of the internal jugular vein, can be heard, according to Parrot,[1] in all persons when lying down.

ii. Systolic murmurs frequently exist at the root of the neck in cases of tricuspid regurgitation. Parrot declares that a double murmur, præsystolic and systolic, can be heard in such cases.

iii. Diastolic murmurs have been described, by Friedreich, as occurring occasionally in the internal jugular. Hypertrophy of the heart,

[1] Etude sur les murmures vasculaires de la région du cou. Arch. Gén. de Méd. 1867, p. 649.

dilatation and strong pulsation of the aorta, and an anæmic state, are the conditions necessary to the presence of this murmur. It is supposed to be due to pressure of the aorta upon the innominata or vena cava, during the diastole. The murmur has been heard in two cases only, one a case of aortic regurgitation, and the other of exophthalmic goitre.

SECTION IV.

EPIGASTRIC PULSATION.

The causes of epigastric pulsation are these: displacement of the heart to the right, the organ becoming vertical; pulsation of the abdominal aorta, or of the cœliac axis, or of an aneurysmal tumour, or of a tumour seated upon the abdominal aorta; regurgitation of blood into the hepatic veins, consequent upon dilatation of the right side of the heart, a phænomenon, first observed by Senac.

The possibility of an epigastric pulsation ever being directly due to the systole of the right ventricle is denied by Hamernjk and Friedreich.[1] However greatly the right ventricle be dilated,

[1] Krankheiten des Herzens. Erlangen, 1867.

it never comes to lie behind the epigastrium. What is commonly regarded as the epigastric pulsation of a dilated heart is no more than a movement conducted to the pit of the stomach from the real place of impulse. Albeit, in these cases, the right ventricle does come nearer to the epigastrium than is natural, and may in fact lie so low as to beat against the xiphoid cartilage.

Recession of the epigastrium, systolic, occasionally simulates pulsation, and when well marked is probably due to pericardial adhesions.

SECTION V.

THE POSITION OF THE DIAPHRAGM.

In many diseases of the chest, it becomes an important element in the diagnosis to ascertain the position of the diaphragm. Strictly speaking, we determine the lower limits of the lungs and heart, the upper limits of the liver, spleen, and stomach, and deduce the position of the diaphragm from these data. For this purpose all the means of physical examination are more or less serviceable, but percussion is especially useful.

¶ I. IN HEALTH.

By inspection, the position which the diaphragm held before puberty may be roughly determined (page 17). By palpation, the position of the heart's apex-beat, and the point where vocal vibration ceases, are ascertained. By percussion, the lung, at the end of an ordinary inspiration, is found to reach, in the sternal line the lower border of the sixth rib, in the nipple line the upper border of the seventh rib, in the axillary line the lower border of the seventh rib, in the scapular line the ninth rib, and in the spinal groove the eleventh rib. The lung, during quiet breathing, never fills the whole pleural cavity, but leaves it unoccupied at the part most distant from the bifurcation of the bronchi, namely, at the semicircular channel formed by the chest wall and the diaphragm where it shelves downwards to be attached to the ribs. This unoccupied portion of the pleural sac has been named the complemental space; there the costal and diaphragmatic pleuræ are in contact.

¶ II. IN DISEASE.

The disease may be such as to afford an obvious impediment to physical diagnosis, by destroying the resonance of the parts above the diaphragm. Which is the case in solidification of the lower lobe of the lung, and in liquid pleural effusion. When the lung is solidified, vocal thrill, if present, will assist us, inasmuch as it fails rapidly beyond the pulmonary region. When liquid is present in the lower part of the pleura, it is impossible to do more than guess where the level of the diaphragm may be. The diaphragm lies high in :— contraction of the lung, distension of the abdomen, paralysis of the diaphragm. The diaphragm lies low in :—hypertrophous emphysema of the lungs, pleural effusions, hypertrophous dilatation of the heart, pericardial effusions, intrathoracic tumours, spasm of the diaphragm. A greatly enlarged heart or an abundant pericardial effusion may depress the diaphragm so much as to produce a tense swelling in the epigastrium : a fact known to Auenbrugger and Corvisart. Depression of the right wing of the diaphragm sometimes depresses the right lobe of the liver in such a

manner that the left lobe is tilted upwards and raises the apex-beat of the heart.

SECTION VI.

THE POSITION OF THE MEDIASTINUM.

The position of the mediastinum is determined in the same manner as the position of the diaphragm, namely, by ascertaining the position of the organ which is most intimately connected with the mediastinum, that is the heart. By palpation we discover the position of the apex-beat of the heart: by percussion we are enabled to confirm the notions acquired by palpation, and to map out exactly the position assumed by the heart: auscultation likewise is serviceable to the same end. The mediastinum is displaced in unilateral pulmonary or pleural disease; and the displacement is either towards or away from the seat of disease. The mediastinum is displaced towards the seat of disease when one lung is shrunken: this is especially seen in phthisis, but also, to a less extent, in an adherent pleura. The mediastinum is displaced away from the seat of disease, in unilateral pleural effusions of liquid or gas. When the effusion is liquid we possess an additional

means of determining the position of the mediastinum, to wit, percussion of the sternal region above the heart. The upper part of the sternum naturally yields a clear resonance: under the pressure of a copious liquid effusion into either pleura, the mediastinum bulges so much towards the unaffected side, as to afford absolute dulness to percussion in the sternal region, and even somewhat beyond it. Intrathoracic tumours also displace the heart.

Dr. Powell[1] has recently shown that the displacement of the mediastinum, which takes place in unilateral pleural effusions, is not wholly due to the pressure exerted by the effusion. The lungs, in health, are in a state of distension which is kept up simply by excess of atmospheric pressure from within; the thoracic walls bearing off the atmospheric pressure from without. The elasticity of the lung is continually striving to overcome this distension, as is manifested by the relaxation of the lung which ensues when the internal and external atmospheric pressures are equalised. The distended lungs of the healthy chest, with their elasticity in full play, drag upon the mediastinum; which,

[1] Notes on the Pneumothorax occurring in Phthisis. London, 1869.

however, maintains its natural position, because the forces on both sides are equal. But if the elastic traction of one lung be destroyed by relaxation or collapse, the other lung, no longer counterbalanced, itself relaxes as much as possible, and draws the mediastinum away from the middle line.

SECTION VII.

THE HEIGHT OF THE PULMONARY APICES.

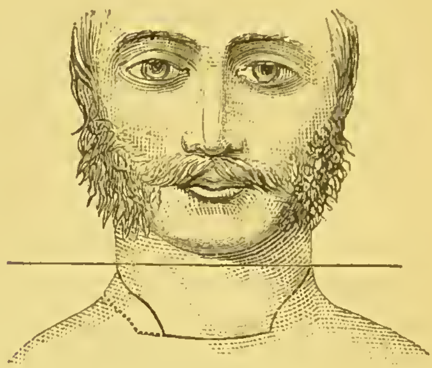
The height to which the lungs reach is determined by percussion of the supraclavicular and suprascapular regions.[1]

In health :—the resonance ceases at a line drawn, from the insertion of the sternomastoid muscle, obliquely upwards to the anterior margin of the trapezius, thence round the muscle, in a line curved with its convexity downwards, to the vertebra prominens. Under the trapezius, the resonance reaches $1\frac{1}{4}$ to 2 inches above the clavicle: the height is mostly equal on both sides, but there may be a difference of half an inch in favour of the right side.

[1] Seitz (autore Heyer): Ueber die percussorische Grenzbestimmung der Lungenspitze. Inaug. Diss. Giessen, 1863. Referred to by Paul Niemeyer: Handbuch der Percussion und Auscultation. Bd. i. 1868.

In disease :—the apex rises unnaturally high, and bulges, in hypertrophous pulmonary emphy-

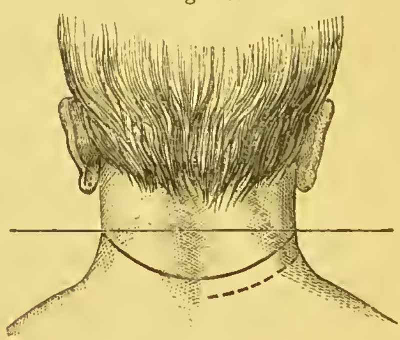
Fig. 16.



HEIGHT OF PULMONARY APICES: (COPIED FROM BOOK OF PAUL NIEMEYER.)

sema: it shrinks, both vertically and transversely, in phthisis. The practical value of de-

Fig. 17.



HEIGHT OF PULMONARY APICES.

termining the size of the pulmonary apices lies in the diagnosis of commencing phthisis.

SECTION VIII.

VASCULAR MURMURS IN THE LUNGS.

Very probable though the occurrence of murmurs within the vessels of the lungs may be, yet twice only has the fact been proved. In the right supraspinous fossa of a phthisical patient, Gerhardt heard, beside bronchial breathing and ringing rales, a systolic whiff; which was explained, post mortem, by the existence of a dilated branch of the pulmonary artery, running across a cavity, and expanded in one spot to an aneurysm the size of a pease.[1] Between the scapulæ and the vertebræ Immermann once heard systolic murmurs, due to constriction of the pulmonary arteries at their entry into the lungs, and just beyond; constriction caused by cirrhotic condensation of the pulmonary tissue.[2] Murmurs of similar character, and presumed to be of similar origin, have been heard by many other persons.[3] There is reason to believe that,

[1] Lehrbuch, pp. 173, 239.

[2] Strictur beider Hauptäste der Lungenarterie und ihrer ersten Verzweigungen in Folge chronischer interstitieller Pneumonie. Deutsch. Arch. für klin. Med. 1869.

[3] Cejka: Beobachtungen über das Nonnengeräusch. Vierteljahrschrift: Prag. Vol. xxvi. 1850. A murmur,

in all cases, the branches of the pulmonary artery constitute the seat of the murmur; which, although most frequently heard in phthisis, may be present also in lung simply collapsed, and in pneumonia.

heard between the upper inner angle of the scapula and the vertebræ, attributed to the underlying veins.

Wintrich: Einleitung, p. 171. A murmur, apparently due to the same cause as that described by Immermann, but heard near the heart in front.

Bartels: Arch. für klin. Med. 1869. Narrates several cases believed to exemplify murmurs generated in the branches of the pulmonary artery.

PART THE SECOND.

CHAPTER I.

PULMONARY CATARRH.

ALL diseases of the lungs tend to be complicated by catarrh. In this place I speak especially of catarrh which is independent of other pulmonary diseases ; catarrh which is idiopathic, or, if secondary, secondary to a general disease.

Respiratory sounds weakened, and rales : these are the signs of catarrh.

I. Weakening of the respiratory sound is general or local : that is to say, the whole of both lungs, or only parts of them, are so affected. i. Local weak breathing is by far the commoner condition ; is due to the presence of mucus in the tubes ; may amount to complete suppression of sound ; is usually moveable in

seat; and is therefore of short duration at any spot. The breath-sound in the unobstructed parts of the lungs is loud or puerile, in proportion to its weakening elsewhere. This supplementary breathing may be so very loud (ultra-puerile) as to hide every other respiratory sound, and to seem universal. ii. General weak breathing is chiefly due to swelling of the mucous membrane.

II. Rales are of two kinds, sonorosibilant and mucous. Both kinds supersede the respiratory sound more or less; that is to say, it is not heard, and, to a certain extent indeed, not produced. Often enough there are no rales at all. i. Sonorous and sibilant rales indicate local incomplete obstruction of the larger air-passages by mucus: a cough, by removing the mucus, mostly removes the rale. ii. Mucous rales indicate the presence of a larger amount of mucus in the tubes. The size of the rale is usually proportionate to the size of the tube wherein the rale is produced. A mucous rale requires that the respiration be fairly vigorous: when the ebb and flow of air is much impeded the rale becomes imperfectly developed (obscure rale): a deep breath will sometimes change an obscure into a distinct rale.

Uncomplicated pulmonary catarrh is eminently bilateral, affecting both lungs: the mucous rales, when present, are most abundant behind and at the bases of the lungs, or indeed exist there only. The percussion-note is often lacking in clearness over parts of the chest, and for a short time, especially in young persons. On the other hand, in both children and adults, patches of unnaturally clear resonance may be met with; probably due to local relaxation of the lung-tissue. In very young children, and in the subjects of rickets, severe pulmonary catarrh is attended by all the signs of inspiratory dyspnœa (page 36), namely, recession of the epigastrium, of the ribs and cartilages below the nipples, and of the supraclavicular spaces. In the same class of patients, vicarious emphysema or insufflation of the lungs is sometimes rapidly developed; it is indicated by bulging of the front of the chest.

When catarrh is complicated by disseminated solidifications of the lungs (especially lobular pneumonia and miliary tuberculosis) the rales sometimes acquire a sharp ringing quality. Persistent localisation of the signs of catarrh to one lung, or to parts of the lungs other than the bases, is an almost sufficient proof that the

catarrh is complicated with some other disease at the part affected.

Œdema of the lungs, blood and diphtheritic exudations in the air-tubes, and miliary tuberculosis, cannot be distinguished from catarrh by the physical signs alone. Pneumothorax is simulated when there is complete catarrhal obstruction to the main bronchus of a lung; an accident sometimes met with in the stupor of cerebral diseases, for example. The breath-sound is suppressed over the whole of one side, and the percussion-note is clear. But in catarrh the suppression of respiratory sound is very transitory, and the displaced mediastinum and amphoric signs of pneumothorax are wanting. Obstruction of a main bronchus by means of a foreign body, a pebble for instance, is much more abiding than catarrhal obstruction, and is moreover attended by rapid retraction of the affected side. Pleuritic friction (friction-rale) is sometimes mistaken for mucous rale: friction of that kind however is mostly unilateral. A certain combination of the signs of catarrh, discovered in a patient examined for the first time, strongly resembles phthisis. This is not uncommon, especially in children and young adults. There are dulness to percussion at one apex,

weak or puerile breathing there, and universal sonorous or mucous rales. The distinction is to be found in the fact that these signs are very transitory when catarrhal, lasting not more than a day or two.

CHAPTER II.

PULMONARY ŒDEMA.

PULMONARY œdema is met with as part of a general dropsical disposition, or as a consequence of failure in the heart's contractions during the agony of dying. I shall speak of the first kind of œdema only.

The physical signs of pulmonary œdema are dependent on the presence of thin liquid in the air-passages, the occurrence of disseminated collapse, and the coexistence of hydrothorax. Respiration weakened up to suppression, and more or less covered by loose mucous rales, with crepitation here and there; these are the most important signs. Percussion-note unaffected; or somewhat diminished in resonance; or unnaturally clear in patches, where the subjacent lung happens to be relaxed, in consequence of the œdema or of the hydrothorax. Dulness to percussion, at the bases of the chest, and bronchial breathing, in proportion to the

amount of pleural effusion. Great inspiratory dyspnœa; attended, it may be, by extreme recession of the infra-mammary regions, even when there is hydrothorax. Acute dilatation and pulsation of the jugular veins. Bronchial respiration sometimes occurs in compact œdema, independently of compression of the lung by exudation into the pleura.

CHAPTER III.

PULMONARY CONGESTION.

THE only physical sign which simple pulmonary congestion is known to produce consists in an intensification of the cardiac second sound over the pulmonary artery; in other words, the pulmonary second sound is louder than the aortic. Great importance has been attributed to this sign by some physicians. In simple congestion of the lungs the pulmonary sigmoids ought, no doubt, to be shut with greater force than the aortic. But the evidence which auscultation has been supposed to give of increased pulmonary tension is, as Dr. Andrew has shown, somewhat fallacious.[1] For greater loudness of the pulmonary second sound may be merely relative, and due to weakness of the aortic sound: or the pulmonary sound may be conducted better than the aortic: or, again, a

[1] St. Bartholomew's Hospital Reports, vol. i. 1865.

pulmonary sound, which is really accentuated, may seem not to be so, because conducted badly.

A few years ago Woillez sought to establish the existence of a form of disease, hitherto misunderstood, and to which he gave the name of idiopathic pulmonary congestion.[1] Unilateral expansion of the base of the chest, discoverable by means of the cyrtometer, is the chief ground of diagnosis : the other physical signs are simply those of catarrh. That pulmonary congestion is the cause of either the bilateral or unilateral enlargement of the thorax in such cases is at present a pure hypothesis : nevertheless the fact remains that such enlargements do occur.

[1] Arch. Gén. de Méd. 1866.

CHAPTER IV.

PULMONARY HÆMORRHAGE.

THE physical signs of a moderate bronchial hæmorrhage are simply rales due to the presence of blood in the tubes : when the blood is expectorated as fast as poured out, obviously there will not even be rales. Hæmorrhagic consolidation of the lung, whether infarctus or apoplexy with laceration, is never sufficiently extensive to yield definite physical signs.

CHAPTER V.

PULMONARY EMPHYSEMA.

BY pulmonary emphysema I mean a progressive degeneration of the lung structure, leading to dilatation of the air vesicles and destruction of the alveolar septa. The size of the lung is mostly increased, sometimes not: hence two varieties.

I. *Hypertrophous Emphysema.*

The diagnosis depends upon the bilateral increase in the size of the lungs, as indicated by: i. Bilateral enlargement of the thorax (page 23): ii. Depression of the diaphragm, involving depression of the heart, liver, spleen, and stomach (page 174): epigastric pulsation is frequently present, due, no doubt, to the low position of the heart (page 171): iii. Encroachment of the lung in front of the heart; whereby the area of superficial cardiac dulness is diminished or abolished, and the heart's impulse and sounds

become enfeebled : iv. Bulging of the lungs above the clavicles.

The respiratory movements tend to assume the characters described under the name of non-expansive inspiration and expiratory dyspnœa. The percussion-note tends to fall in pitch, that is to say, to become tympanitic ; the muffling mostly remains unchanged, or is even increased, although sometimes the note becomes clear in places. The respiratory sound is usually weakened, but may be slightly bronchial here and there. Friction sound is occasionally produced by distended subpleural sacculi (page 131).

Emphysema is always associated with pulmonary catarrh, and the physical signs are modified accordingly. Dilatation of the heart is a necessary consequence of long-standing emphysema.

Pneumothorax is the only disease which can possibly be confounded with emphysema. But bilateral pneumothorax is incompatible with life, and emphysema always is bilateral. Moreover the amphoric signs are never present in emphysema.

II. *Atrophous Emphysema.*

Atrophy of the lungs with dilatation of the air vesicles, occurs as a part of general senile atrophy, or as a consequence of previous pulmonary disease, especially arrested phthisis. The physical signs are those of hypertrophous emphysema, excepting the signs indicative of increased size of lung; usually however the lung does encroach upon the præcordial region, and supra-clavicular bulging is sometimes present. Tympanitic percussion-note (which may be very muffled) is always audible over a considerable surface of the chest; add to this, the absence of definite signs of other disease; and these are all the data we possess for the diagnosis of atrophous emphysema in any case of which the symptoms point, without doubt, to disease of the lungs.

CHAPTER VI.

PULMONARY COLLAPSE.

COMPLETE collapse of large portions of lung occurs as a consequence of compression or catarrh. Pleural effusions (liquid or air), pericardial effusion, hypertrophy of the heart, and intrathoracic tumours, compress the lung so as to empty it of air. The physical diagnosis can have reference only to collapsed lung in contact with the chest wall; and even then the signs are not very distinctive; dulness to percussion, persisting vocal fremitus, and bronchial breathing. When only a small portion of lung is collapsed, it is usually surrounded by relaxed pulmonary tissue, which gives a clear, high-pitched resonance. Increase of the superficial area of cardiac dulness is a conceivable event, when the anterior parts of the left lung are collapsed.

Collapse of a few vesicles is indicated when deep inspiration brings out a crepitant rale audible for two or three breaths only, and then heard no more.

The diagnosis of congenital atelectasis receives small help from physical examination, unless there be marked inspiratory dyspnœa with recession of the chest-walls.

CHAPTER VII.

PLEURISY.

ALTHOUGH pleurisy cannot exist without inflammatory effusion, yet the phrase, pleurisy with effusion, has come to be applied to those cases only in which the effusion is considerable; these will be discussed hereafter. In the present place I purpose to treat of pleurisy attended by comparatively small effusion.

When pleurisy of this kind is universal, it strongly simulates phthisis. The affected side is retracted, it may be considerably, and moves much less freely than in health. The percussion-note is raised in pitch and muffled, over the greater part or the whole of the side. The sense of resistance is increased. When the disease affects the left side, the superficial area of cardiac dulness is extended. The respiration generally is weak, and attended by a friction-rale indistinguishable from the mucous rales of

phthisis. At places the breath-sound may be bronchial, in all degrees of intensity, up to a hollow resonance such as Laennec himself would have supposed to leave no doubt of cavity. Add to the physical signs hectic fever, and we cease to wonder that pleurisy of this kind is usually mistaken for phthisis more or less advanced. However the pleuritic patients recover completely, without a vestige of disease left behind, save haply a slight unilateral retraction of the chest. Whenever the signs of a case of supposed phthisis are in some respects peculiar; whenever they indicate advanced disease limited to one side of the chest; whenever cavernous signs are heard in unusual places; it is well to weigh the possibility of simple pleurisy. Careful microscopic examination of the sputa will then help us where physical examination fails.

Much less severe forms of pleurisy, accompanied by local diminution of resonance and by friction sounds, are often met with: the diagnosis depends upon the friction sound, and, where that is undecisive, upon collateral circumstances.

CHAPTER VIII.

PLEURAL EFFUSION.

THE different kinds of effusion into the pleura may be thus classified :—

1st. Gaseous		. . .	Pneumothorax.
2nd. Liquid :	i. dropsical	.	Hydrothorax.
	ii. inflammatory		Pleurisy with effusion.
	iii. blood	. .	Hæmothorax.

The effusion is either unilateral or bilateral : either total (= filling the whole of the pleural cavity) or partial : and a partial effusion is either loculated (= enclosed in adhesions) or free. The lung collapses in a degree proportionate to the abundance of the effusion. Wherefore total pleural effusion is always unilateral, inasmuch as collapse of both lungs is obviously incompatible with life.

CHAPTER IX.

PNEUMOTHORAX.

THE varieties of pneumothorax are these :

1st. Air and liquid present in the cavity :	Hydro-(pyo-)pneumothorax.
i. Cavity large :	
α. closed.	
β. with an external fistula	(fistulous empyēma.)
ii. Cavity small . . .	Loculated Pneumothorax.
2nd. Air alone present in the cavity .	Pure Pneumothorax.

I. *Closed Hydropneumothorax.*

Its physical signs are these :—unilateral distension of the chest, tympanitic percussion sound, enfeebled respiration, and amphoric phænomena.

1st. Distension of the chest is indicated by :— i. Unilateral enlargement (page 28); sometimes so great as to cause an excess of three inches in the semi-circumference on the affected

side. ii. Depression of the diaphragm (page 172); sometimes so great as to force the upper surface of the liver altogether below the level of the costal margin in front, and to produce a band of tympanic resonance in the abdomen, above the liver dulness. iii. Displacement of the mediastinum towards the unaffected side (page 175); at first due no doubt to traction exerted by the lung which is not collapsed; but before long the air in the pneumothorax comes to exert pressure upon the mediastinum.

2nd. The percussion-sound falls in pitch and increases in duration, that is to say, becomes tympanitic, in proportion to the distension of the pleura. The note, however, remains muffled; and, in fact, when the distension is extreme, the muffling approaches dulness (page 76). Amphoric quality is sometimes possessed by the percussion note, sometimes not. Where liquid is present, there, of course, non-resonance will be found: the liquid effusion is free, moveable, and changes its position along with change in the position of the body.

3rd. The respiratory sound is weakened in proportion to the collapse of the lung. Sometimes the collapse is so complete, that no breathing is audible, except in the vertebral

grooves. When the lung has been much solidified by previous disease, so that collapse can hardly ensue, a respiratory sound, more or less loud and bronchial, will be heard all over the pneumothorax.

4th. Amphoric signs, indicative of a large cavity, are present. i. Amphoric echo may attend the sounds of breathing, coughing, or talking. And let it not be supposed that amphoric respiration is necessarily due to air passing freely out of the lung into the pleural cavity and back again : on the contrary, this is rarely the case; the breath sounds heard are produced in the lung, and acquire their amphoric quality merely by transmission through the pneumothorax. ii. Metallic tinkling may be present. iii. The bell-sound is a very important sign of a large pneumothorax. iv. Succussion splash occurs when the quantity of liquid is considerable.

The inspiratory movement of the affected side is non-expansive (page 37) : the vocal thrill is diminished, or, haply, abolished. The respiration on the unaffected side is puerile. When the pneumothorax is secondary to phthisis, the apex of the lung often remains adherent. In an old pneumothorax a large ulcerous opening

is sometimes formed between the cavity and a large air-tube : thereupon, all active distension of the side ceases. In rare cases air is effused into the pericardium as well as into the pleura, whereby the signs indicative of a displaced mediastinum are lost.

II. *Fistulous Empyema:*

Differs from a closed hydropneumothorax in that the affected side is distended very slightly, or not at all, or more frequently is contracted. The sound of lung-shock is occasionally produced in a fistulous empyema (page 131).

III. *Loculated Pneumothorax :*

Is not uncommon ; and when associated, as it often is, with a permanent pulmonary fistula, the diagnosis from a cavity formed within the lung becomes difficult, or even impossible. A large intra-pulmonary cavity will often produce bulging of the chest walls ; whereas a loculated pneumothorax is mostly surrounded by adhesions which are very dense and indistensible.

IV. *Pure Pneumothorax :*

Is a rare condition, mostly of traumatic origin, but occasionally due to rupture of distended pul-

monary air-vesicles. The physical signs are the same as those of a closed hydropneumothorax, excepting such as are due to the presence of liquid.

CHAPTER X.

HYDROTHORAX.

HYDROTHORAX and œdema of the lungs, pleural and pulmonary dropsy, own the same causes, and therefore mostly coexist. A serous effusion due to pleurisy often obtains the name of hydrothorax; but the word is used in this place to signify a simple dropsy.

Hydrothorax is bilateral, although, on account of local influences, the quantity of effusion is not always equal on both sides. And, being bilateral, the fluid cannot occupy more than a portion of the pleural cavities; the effusion is partial. When pulmonary œdema also is present, it is possible for great inspiratory dyspnœa (page 36) to concur with copious hydrothorax. The diaphragm is depressed: the base of the thorax expanded: the position of the mediastinum remains unchanged.

Dulness to percussion coextensive with the effusion. The liquid effused, not being enclosed in adhesions, is freely moveable in the pleura,

and always occupies the most depending part: upper limit of dulness, for the same reason, horizontal. Vocal thrill diminished, respiratory sound and vocal resonance either simply weak or feebly bronchial, over the regions where the percussion note is dull.

CHAPTER XI.

PLEURISY WITH EFFUSION.

PLEURISY with effusion is usually unilateral. When bilateral, it is mostly secondary to a general septic or putrid state, and rapidly proves fatal. I shall divide the course of pleurisy, according to scholastic terms, into periods of increase, height, and decline.

I. Period of increase. Sometimes a friction sound is the earliest sign of pleurisy with effusion : and when this is so, the sound commonly possesses the character of a friction-rale (page 129), and is heard over the whole of one side of the back. More frequently, however, the earliest signs are those of a liquid effusion : the same signs, when they have been preceded by a friction sound, rapidly supersede it. The liquid tends to collect, as soon as formed, in the lowest place. At first, when the quantity is small, the lung is simply relaxed, and swims upon the effusion : but as the liquid accumulates

it exerts pressure upon the lung, and renders it more or less empty of air.

The great sign of liquid effusion is a co-extensive dulness to percussion. This dulness begins at the lowest part of the chest behind; the note being natural elsewhere. When the effusion rises higher than the angle of the scapula, the lung will have become relaxed to such an extent as to give a clear subtympanitic or trachæal note above the nipple of the same side in front. Whether, by further increase in the quantity of the fluid, the whole back become dull before the front is so at all, or whether the upper level of the fluid be comparatively horizontal, depends wholly upon this circumstance, namely, whether the patient took to his bed early in the disease, or whether he kept about while the effusion was going on. The level of dulness is always higher behind than before. The line of demarcation between resonance and non-resonance is sharper before than behind. The anterior clear resonance, when present, is sometimes of cracked-pot quality.

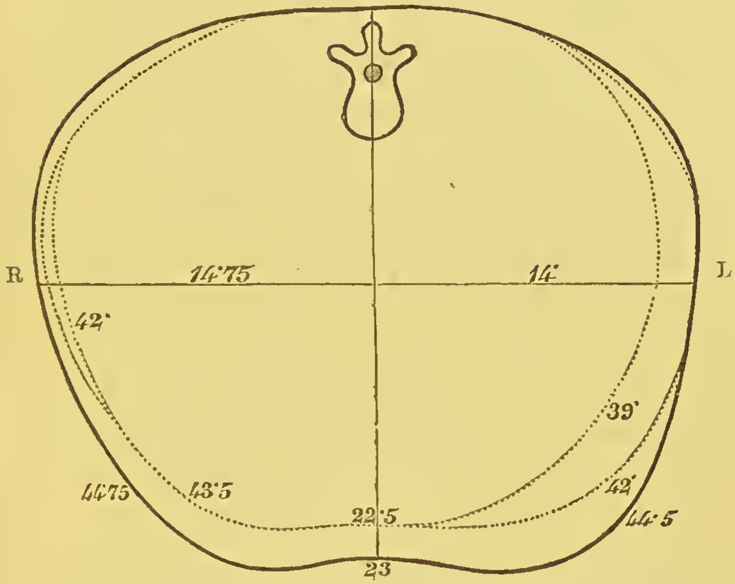
In proportion to the amount of effusion, the side is enlarged (page 28), the diaphragm depressed (page 172), and the mediastinum displaced (page 175). The vocal thrill is dimi-

nished where dulness to percussion exists, and is wholly abolished in great distension of the side. The respiration, at first weakly vesicular, soon becomes bronchial: and usually, with progressive increase of effusion, the bronchial breathing becomes less and less loud until, at last, it may be wholly suppressed. But sometimes, although the quantity of fluid be very great, loud bronchial breathing is heard all over the affected side: the fact being that the loudness depends, not inversely upon the quantity of fluid effused, but directly upon the amount of lung left permeable to air. Breathing intensely bronchial, large mucous rales, and pectoriloquy, might lead the unwary into the diagnosis of phthisis:[1] that the respiratory sound should ever be attended, in simple pleurisy with effusion, by an amphoric echo, such as is heard in pneumothorax, is, I must confess, hard to believe. Vocal resonance weak and bronchial in much the same manner as the respiratory sound. When the effusion is partial, with clear resonance in front, the bronchophony is usually ægophonic about the angle of the scapula.

[1] Monneret: Gaz. Médic. 1842. Rilliet et Barthez: Arch. de Méd. 1853. Béhier, ibid. 1854. Landouzy, ibid. 1856.

II. The effusion at length reaches its height. This sometimes will not be until the pleural

Fig. 18.



COURSE OF PLEURISY WITH EFFUSION, LEFT SIDE.
Outer line = horizontal section before paracentesis.
Middle line (dotted) = four days after paracentesis.
Inner line (dotted) = three weeks after paracentesis.

cavity is tensely full; or the effusion may stop at any point short of that extreme. When the quantity of fluid on the left side is very great, the left half of the diaphragm is occasionally depressed to such an extent that not only can the lower margin of the spleen be felt, but even its upper margin, in fact its whole circumference.

And at the same time, tho thrusting of the heart and mediastinum over into the right side of the thorax may cause the right wing also of the diaphragm to be depressed to an almost equal degree ; a point ascertained by examination of the liver. When the effusion is partial it does not shift with the position of the body, as a dropsy of the pleura does ; the fluid of an inflammatory effusion is enclosed in more solid exudation. The semicircumference is sometimes actually less on the diseased than on the healthy side.

III. Period of decline. When a pleural effusion undergoes absorption, the following series of physical signs are noted.

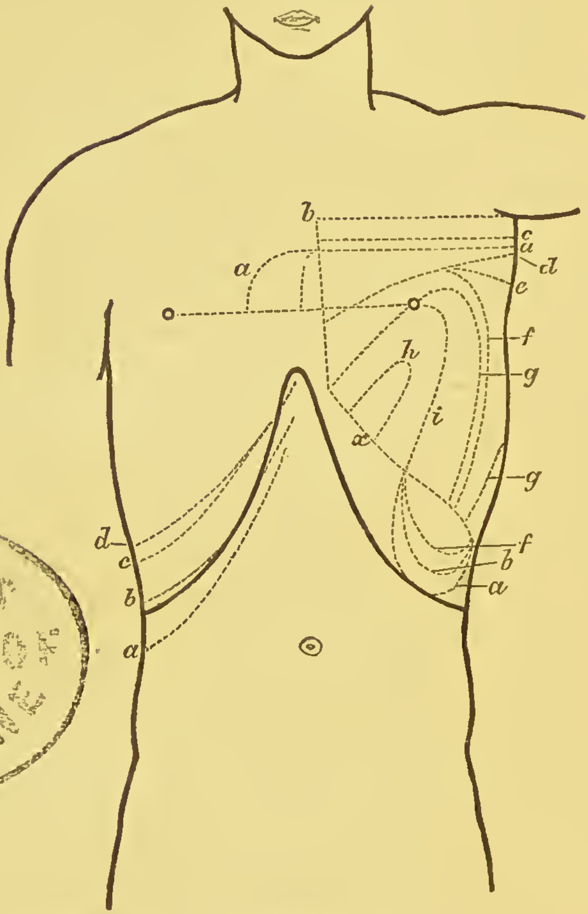
The diaphragm and mediastinum go back towards their natural position : to follow the retreating organs is the best means of marking the progress of absorption, whilst the quantity of the effusion remains great. The distension of the affected side becomes less ; and accurately to register this fact is a most important service rendered by the cyrtometer. When the effusion has so far diminished that the lung again comes into contact with the chest-wall, percussion enables us to follow the falling level of fluid. And, at the same time, auscul-

tation will inform us when and where actual adhesion of the opposed surfaces of the pleura has occurred.

With reference to the percussion signs more particularly. Dulness, only just short of absolute, often remains for a considerable time after perfect adhesion has occurred. This fact, and another before alluded to, namely, that the limit of absolute dulness cannot be always sharply defined posteriorly, sometimes render the attainment of our aim in percussion difficult. Nevertheless a distinction between dulness which is absolute and that which is not, can usually be made by careful superficial percussion: and the difficulties, which I have spoken of, are by no means present in every case. The manner in which the effusion is absorbed (like the manner in which it is effused) depends upon circumstances, whether the patient is able to keep upright during his illness, or not. In the former case, the course of a decreasing effusion has been carefully studied by Damoiseau.[1] The upper surface of the liquid, when it reaches as high as two inches above the nipple, is horizontal: when lower than this point, the dulness

[1] Recherches sur quelques points du diagnostic des épanchements pleurétiques. Arch. de Méd. 1843.

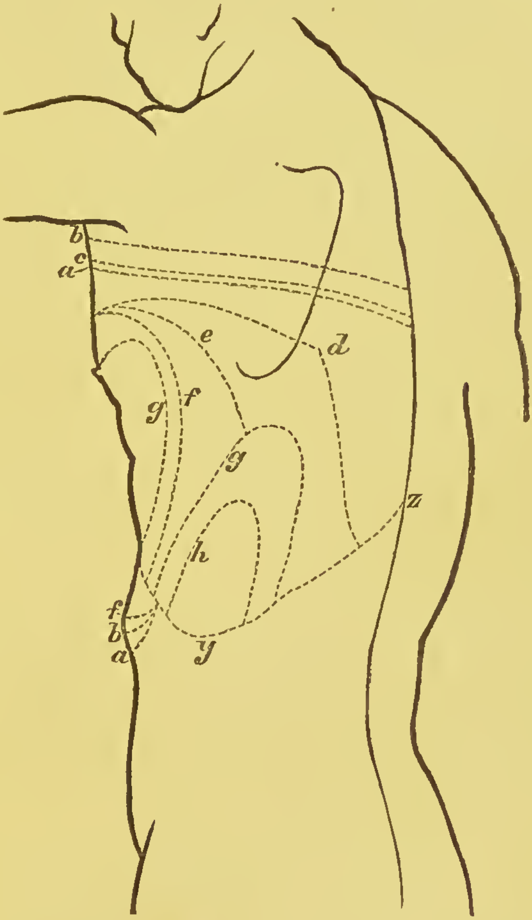
Fig. 19.



THE COURSE OF AN EFFUSION INTO THE LEFT PLEURA: FROM DAMOISEAU.

a a = limits of effusion, heart, liver, and spleen, when patient first seen.
b b = same on first day of treatment. *c c* = second day.
d d = third day. *e e* = fourth day.
f f = fifth day. *g g* = morning of sixth day.
i = evening of sixth day.
h h = last limits of effusion previous to disappearance, *i c.*, on tenth day.
x y z = lower limit of pleura.

Fig. 20.



COURSE OF AN EFFUSION INTO THE LEFT PLEURA: FROM DAMOISEAU.

forms irregular parabolic curves, which become smaller and smaller, and last of all disappear at the lowest parts of the thorax. The fluid is mostly absorbed in the following order: from the vertebral groove near the root of the lung; from the supramammary region; from the rest of the vertebral groove and infrascapular region; from the inframammary region, and lastly from the lower lateral region; concerning which point it is important to remember that the lowest part of the pleural cavity, in the upright position, is in the axillary line. The curve of the sinking fluid is sometimes double, as happened to be the case with the patient represented in the drawing.

Disappearance of absolute dulness at any spot is nearly always attended, for a day or two, by friction sound, indicative of restored contact between the pleural surfaces: redux friction, as it is usually called.

The two diseases from which it is most difficult to distinguish pleural effusions are cancerous and hydatid tumours within the chest. Cancer of the lung causes no enlargement of the affected side, and the dulness of cancer does not usually follow the laws which have been explained with regard to effusion into the pleura.

Hydatid tumours are uncommon, and hardly admit of diagnosis until the peculiar membrane is expectorated, or removed during the operation of paracentesis.

CHAPTER XII.

EMPYEMA.

AN empyema of the whole of one pleural cavity affords physical signs identical with those which have been described under the head of pleurisy with effusion. Often enough the distension of the affected side is anything but great: the heart for instance may be scarcely at all displaced, a circumstance which is sometimes due to pleural adhesion over the pericardium. An empyema which has been partially discharged so as not to fill the pleural sac, or, what is the same thing, a pneumo-empyema, is characterised by percussion dulness which shifts with the position of the body; inasmuch as there are no adhesions which everywhere limit the effusion, they have all melted down into pus.

But small collections of pus in the pleura are sometimes enclosed in dense adhesions: loculated empyemata. When superficial they will be discovered by the absolute dulness to percussion, weak bronchial breathing, and possible

bulging of the part. When more deeply seated, or when diaphragmatic, diagnosis is impossible. A large diaphragmatic loculated empyema closely resembles an enlarged liver as to the physical signs.

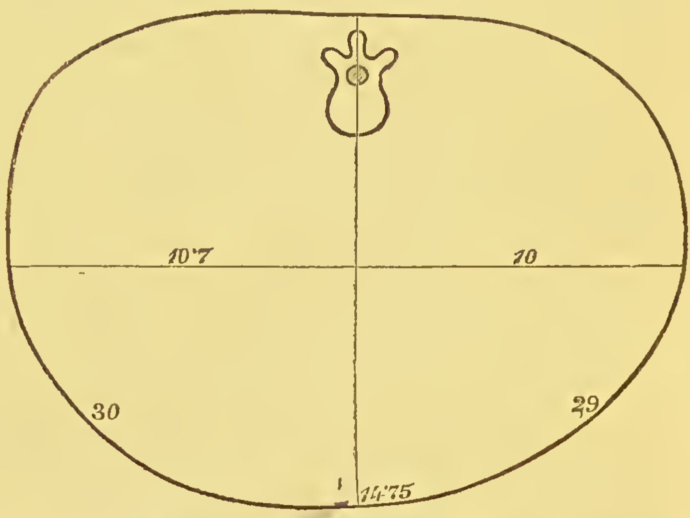
Excessively abundant empyematous effusions sometimes pulsate rhythmically with the heart: pulsating empyemata. The most frequent seat of the pulsation is the anterior part of the chest around and above the nipple.

CHAPTER XIII.

ADHERENT PLEURA.

VERY densely adherent pleuræ are attended by all the signs of unilaterally contracted chest (page 31). The percussion sound will be

Fig. 21.



UNILATERAL RETRACTION OF LEFT SIDE OF CHEST CONSEQUENT UPON AN ADHERENT PLEURA.

deficient, and the breath sound enfeebled, over a large surface of the affected side.

But much looser adhesions, such as are so often unexpectedly found post mortem, and which influence the percussion and auscultation of the chest in no respect, may be sometimes (perhaps always) discovered by the cyrtometer, when one pleura only is obliterated. A proof of this is afforded by the tracing annexed, which was taken from a child who died after an operation, and in whom no symptoms had existed to raise a suspicion of the universally adherent pleura on the left side.

When, by a deep inspiration, the area of superficial cardiac dulness is not diminished, the pleural surfaces over the pericardium are probably adherent.

CHAPTER XIV.

PNEUMONIA.

FOR present purposes it is convenient to divide acute pneumonia (peripneumonia) into two kinds, the lobar and lobular, the consolidation being, in the former case massive, and in the latter disseminated.

I. *Lobar Pneumonia.*

The physical signs differ according to the different stages of pneumonia described by the morbid anatomist.

Stage I. Active sanguineous fluxion upon the lung is characterised by weak respiratory sound, and crepitant rale. The percussion sound is not materially changed : sometimes the note becomes clearer, and, at the same time, high-pitched, a circumstance which is probably due to partial relaxation of the lung-tissue. The duration of this stage is so short that the majority of patients have entered upon the next stage before they are seen by the physician.

Stage II. Hepatisation of the lung is characterised by dulness to percussion, bronchial breathing, and bronchophony. In persons whose chest-walls are resilient, the dulness will be found less absolute than is afforded by pleural liquid effusion, cancer of the lung, or dense phthisical consolidation. When, as is sometimes the case, islets of unsolidified lung are imbedded in the surface of the hepatised tissue, they afford the clear resonance of relaxed lung, or even a cracked-pot sound. A cavity, or a large bronchus, separated from the surface by a thin layer of pneumonic lung, will produce the same effects. The bronchial breathing of pneumonia differs from that usually met with in pleurisy by being more whiffing and high-pitched, tubular in short. Sharp, resonating mucous rales are sometimes present : occasionally friction-sounds. Vocal fremitus natural or increased ; very rarely absent, and only when the inflammatory swelling of the lung is unusually great. All breathing sound and vocal resonance may be absent over hepatised lung : this condition is either transient or permanent ; when transient it is due to obstruction of the tubes by mucus, which a cough can remove ; when permanent, the air-passages contain solid exudation or coagula. The respi-

ration around the fluxionary or hepatised portions of lung is puerile; hence puerile breathing sometimes precedes crepitation or bronchial breathing, when the area of pneumonia is extending. In infants the bronchophonic cry is often the only auscultation-sign of pneumonia which can be obtained. Wintrich[1] and Ziemssen[2] have shown, the former with reference to adults and the latter to children, that, in lobar pneumonia of the lower half of a lung, the chest, on that side, is expanded to the state of deep inspiration. I have already indicated how much this expansion is less than that of a pleural effusion (page 23); moreover, pneumonia never displaces the heart or the mediastinum. The state of deep expiration is the best for discovering the permanent inspiratory expansion of pneumonia.

Stage III. The hepatisation mostly undergoes resolution, which is characterised by progressive diminution of the bronchial quality of the breathing, by the occurrence of mucous rales, and by gradual restitution of the pulmonary percussion-note. Occasionally the solidified

[1] Einleitung, page 84.

[2] Pleuritis und Pneumonie im Kindesalter. 1862, page 235.

tissue softens rapidly down into an abscess, a condition which does not admit of diagnosis. When the inflammatory tissue neither resolves nor suppurates, it undergoes changes which will be described under the head of phthisis, namely, cirrhosis and cheesy degeneration.

Pneumonia of the apex, a disease far from uncommon, is to be distinguished from phthisis by the general symptoms, and by the course of the disease.

II. *Lobular Pneumonia.*

Acute pulmonary catarrh, occurring in children, old people, or debilitated persons, is very apt to be accompanied by lobular pneumonia. Chronic lobular pneumonia is the most important element in many cases of phthisis, and will therefore be described along with the latter disease. In the acute form of lobular pneumonia the catarrh is always severe : consolidation may be suspected wherever the rales are unusually sharp and resonant ; at the anterior margins of the lung, where the pneumonic foci often become confluent, I have occasionally detected dulness to percussion.

CHAPTER XV.

PULMONARY TUBERCULOSIS.

PULMONARY tuberculosis, when developed abundantly and rapidly, presents physical signs or not, according as it is accompanied by pulmonary catarrh or not. A most copious formation of discrete miliary tubercle is often found in lungs which during life afforded no signs of disease. And when signs are present, they are no other than those of universal pulmonary catarrh ; weak respiration, covered by small mucous rales, which are sometimes very sharp and resonant, sometimes not. Although percussion and auscultation fail, yet the whole history of the symptoms mostly yields the means of diagnosis.

CHAPTER XVI.

PULMONARY PHTHISIS.

BY pulmonary phthisis is meant chronic pulmonary consolidation which tends to go on to ulceration. But I shall not follow Bayle [1] in his strict application of this definition; cancerous phthisis it will be best to discuss in a chapter apart. Like acute pneumonia, so phthisis differs in its physical signs according as its consolidations are miliary and disseminated, or massive and involving large tracts of lung. Disseminated miliary nodules which are phthisical do not admit of discovery more easily than those which are tubercular or peripneumonic: the attendant signs are due simply to catarrh.

The physical signs of phthisis depend upon these particulars: the consolidation itself; diminished bulk of the solidified part; the localisation of rales in the solidified part; and the formation of cavities.

[1] "Conséquent à ses principes jusqu'à la sévérité." Laennec: Ausc. Méd. ii. 342.

i. Consolidation is indicated by diminution of percussion resonance; the pitch of the note rises and its clearness diminishes until, in some cases, absolute dulness is reached. In the earlier stage of progressive solidification the respiratory sound is simply weak; later on it becomes more and more bronchial. The bronchial breathing, which at first is due wholly to consolidation of the alveolar structure, subsequently becomes intensified by the formation of cavities. In rare cases, when the solidification is very dense and massive, and not yet excavated, the respiration ceases to be audible at all.

ii. Diminution in the bulk of the solidified part is attended by contraction of the corresponding region of the chest. When the apex of a lung is affected, the signs of a shrunken apex will be present (page 178). When the left upper lobe is affected, the superficial area of cardiac dulness will be increased. It is that form of phthisis called cirrhosis which affords the most marked degrees of shrinking of the chest: [1] in these cases displacement of the medi-

[1] The name "cirrhosis" was first applied to this condition by Corrigan (Dubl. Journ. Med. 1838), but, under other names, this callous form of phthisis has been known to physicians for centuries.

astinum and of the heart, and elevation of the diaphragm occur.

iii. The fixed localisation of mucous rales, in a limited portion of lung, is an important sign of phthisis. At first more or less obscure in proportion to the weakening of the respiration, the rales gradually become clear and resonant as the consolidation and ulceration proceed.

iv. The diagnosis of a cavity demands that it be near the surface, not smaller than a walnut, and containing for the most part air. Under these conditions the formation of a cavity is sometimes attended by alteration of the previously dull percussion note into a clear resonance, of a pitch, which is high in proportion to the size of the cavity, and which is variable according to the openness of the mouth, the more open the mouth the higher the pitch. A large cavity is sometimes attended by a bulging of that part of the chest-wall which had previously been sunken. The bronchial quality of the respiratory sound is always well marked in an empty cavity : the rales are large and resonating. Practically the physical diagnosis of excavation mostly comes to this, that, in advancing phthisis, a cavity is presumed to be present where the bronchial breathing is most

intense. Very large cavities afford amphoric percussion-note, amphoric echo, metallic tinkling, and splashing; but never the perfect bell sound. Having ended this general survey of the signs of phthisis I will now speak of a few additional matters.

The chest of persons predisposed to phthisis is usually phthinoid; and that of persons actually phthisical, flat. Inasmuch as phthisis rarely involves both lungs to an equal extent, unilateral retraction of the chest is mostly present. A much more local shrinking usually occurs where the phthisical processes are most advanced. Occasionally the thorax is of emphysematous shape: this is the case when phthisis is engrafted upon emphysema, and when retrograde phthisis is accompanied by advancing degenerative atrophy of the lungs. A cracked-pot sound is sometimes elicited by percussion over phthisical consolidation, both when a cavity is present and when it is not. A clear high-pitched resonance is sometimes producible in incipient phthisis, when there is no reason to suspect a cavity; superficial relaxation of the lung is probably present in such cases. Sounds other than obvious rales, creaking and rubbing sounds, are not uncommon, and are most likely produced in

the solidified tissues themselves: sometimes, however, undoubted transitory friction is heard. Genuine pneumonia, when it occurs around a phthisical portion of lung, is commonly indicated by increase of the dulness to percussion, and a crepitant rale. When pneumonia occurs around a cavity, which had been previously separated from the chest-wall by aërated tissue, a clear percussion resonance, with pitch variable by opening or shutting the mouth, will be found so long as the pneumonic consolidation remains. Collapse rale may be heard over phthisical lung. The coexistence of a diffused pulmonary catarrh, of emphysema, or of laryngeal disease, is a great impediment to the physical diagnosis of phthisis. Generally speaking, the physician should examine a patient, supposed to be phthisical, several times before giving a positive opinion.

An aneurysmal dilatation of a branch of the pulmonary artery, contained in a phthisical cavity, has been known by Gerhardt to give rise to a shrill systolic murmur audible in the suprascapular fossa (page 179). Murmurs produced in the subclavian artery are not uncommon; they have been already described (page 158).

The respiration in the unaffected parts of the

lungs is puerile, provided it be not modified by the presence of catarrh. The loudness of the supplementary breathing sometimes leads the inexpert to predicate the existence of disease just in that solitary part where the lung remains healthy.

The difficulty of diagnosis between phthisis and some forms of pleurisy and of pneumonia has been already indicated. When mediastinal tumours exert pressure upon the root of a lung, that is to say, upon the pulmonary plexus and bronchi, the tissue of the lung occasionally breaks down into suppurating cavities.[1]

[1] Gull: Guy's Hospital Reports, 1859. Gairdner: Clinical Medicine, 1862.

CHAPTER XVII.

PULMONARY CANCER.

WHAT has been said of peripneumonia, tubercle, and phthisis, is true of cancer; that miliary disseminated consolidation cannot be distinguished by physical examination from simple catarrh. The signs of massive cancer are, in general, absolute dulness to percussion, diminished vocal fremitus, and weak or absent breath-sound. When a large open bronchus is intimately connected with the cancerous mass, bronchial breathing will be heard. Cancerous solidification of the apex of one lung simulates phthisis, especially when the cancer breaks down, as it sometimes does, into cavities.[1] The physical diagnosis in such a case becomes possible only when the cancer begins to involve the mediastinum, or to spread in any other manner unlike the phthisical process. Very large cancerous tumours of the lung afford signs which

[1] J. R. Bennet: Lumleian Lectures on Intrathoracic Cancer. Brit. Med. Journal, May and June, 1870.

for the most part closely resemble those of pleurisy with effusion. But cancer, unless of most rapid growth, does not enlarge the chest, may even cause it to be retracted. The mediastinum and diaphragm are usually not displaced. Nevertheless a quickly-growing tumour sometimes shows a disposition to sprout in different directions, so as to displace the heart or depress the diaphragm. The most useful means of distinguishing between cancer and pleural effusion is drawn from the fact that the distribution of the percussion dulness differs in the two diseases. When dulness does not begin at the bottom of the chest; when there is a great extent of absolute non-resonance in front, and none behind; when, in the midst of a great extent of dulness, we detect one or more small isolated patches of resonance (haply quite clear or even cracked-pot); we may infer the existence of a solid tumour. The concurrence of mediastinal cancer, or of pericardial or pleural effusions, with pulmonary cancer, need not be dwelt upon in this place.

CHAPTER XVIII.

PULMONARY HYDATIDS.

ECHINOCOCCUS cysts, as to their physical signs, resemble, in part solid tumours of the lung, and in part liquid pleural effusions. A deep-seated cyst, even if large, cannot be detected by physical examination. When superficial, dulness to percussion, weak or absent breath sound, and temporary friction sound will be present. Large tracts of lung may be relaxed, just as in pleural effusion, and produce a resonance unnaturally clear. When the tumour becomes of very great size, all the signs of unilateral enlargement of the chest, and of displaced diaphragm and mediastinum occur. Last of all there may be a local hemispherical swelling: this is characteristic. A cyst, which has discharged its contents into the air-passages, may, it is said, afford amphoric signs. And occasionally a hydatid tumour breaks both into the

bronchi and into the pleura: when this accident occurs, all the signs of hydropneumothorax follow.[1]

[1] Case quoted from Mercier by Trousseau: Clinique Médicale, 1865, i. 711.

CHAPTER XIX.

DILATATION OF THE BRONCHI.

BRONCHIECTASIS is usually associated with cirrhotic contraction or with emphysematous enlargement of the lung. The physical signs afforded by a sacculated bronchus are almost identical with those of a phthisical cavity. The diagnosis depends upon collateral circumstances. The conditions requisite for the discovery of a bronchial sac by percussion and auscultation are these : that the cavity be of a certain size, near the surface, surrounded by condensed lung, and containing air as well as liquid. Rapid change in the physical signs, consequent upon profuse expectoration, is a very important evidence of dilated bronchus.

CHAPTER XX.

STATE OF THE LUNGS IN ACUTE DISEASES.

IN all acute diseases Woillez finds that both sides of the chest are enlarged from the very first.[1] He attributes this enlargement to an active hyperæmia of the lungs. It would seem more probable that, owing to increased depth and frequency of respiration, the chest is maintained in a state of fulness approaching that of the deepest inspiration.

[1] Mémoires de la Soc. Méd. d'Observation, vol. iii.

CHAPTER XXI.

ASYSTOLISM.

ASYSTOLISM is a useful word, employed by Beau,[1] to designate that remarkable group of symptoms which is characteristic of an enduring inability in the right ventricle to empty itself. The physical signs attendant upon asystolism are those of dilatation of the right side of the heart. A short time before death additional signs occur. i. Great weakening of the impulse. ii. Great weakening of the heart-sounds, and of any murmurs which may have been present, until at last nothing is heard save a kind of humming. iii. A systolic murmur, best heard at the inner part of the fourth left interspace, and due to regurgitation through the tricuspid orifice, frequently occurs when asystolism becomes urgent, but before the heart is so weakened as not to be able to produce a

[1] Beau: Considérations générales sur les maladies du cœur. Arch. Gén. de Méd. 1853.

murmur.[1] Should the symptoms of asystolism moderate, the murmur disappears.

[1] Parrot: Etude sur un bruit de souffle cardiaque symptomatique de l'asystolie. Arch. Gén. de Méd. 1865.

CHAPTER XXII.

ENLARGEMENT OF THE HEART.

TWO lesions, rarely separated, go to make up enlargement of the heart, namely, hypertrophy of the muscular substance, and dilatation of the cavities. But the proportion in which the two elements coexist differs in different cases. An enlargement, due more to hypertrophy than dilatation, was named by Corvisart an active aneurysm of the heart: the reverse condition he called a passive aneurysm: the words active and passive used in this sense are very useful. Although hypertrophy and dilatation are associated in fact, it is easy to dissociate them in idea, and to consider the pathology of each apart. Moreover this it is necessary to do, inasmuch as they are, in nearly every particular, antagonistic. I shall therefore treat of the signs, first of hypertrophy, and next of dilatation.

Enlargement of the heart may be simulated by pericardial effusion; by intrathoracic tumour

(aneurysmal or not), above the base of the heart, or pushing the heart forwards against the front of the chest; by mere increased extent of contact between the heart and the chest-wall; by consolidation of the anterior part of either lung; and by a pleural effusion.

CHAPTER XXIII.

HYPERTROPHY OF THE HEART.

THE sign of hypertrophy of the heart is a heaving impulse. Not that a heaving impulse is always present in every case of hypertrophy; feeble action of the heart, degeneration of its texture, and other conditions tend to counteract the heaving impulse; in its absence however there is no other certain sign of hypertrophy. The impulse, because increased in power, is increased in extent also. When the right ventricle is hypertrophied, the conducted epigastric impulse is strong. When the left ventricle is hypertrophied, the apex of the heart reaches farther to the left than natural, in consequence of the elongation of the aorta produced by the hypertrophy.

The powerful action of the heart causes the sounds to be loud and clear. The arterial pulse is forcible and visible, except when the aortic orifice is constricted, or when the vigour of the heart fails. A very sharp whizz is heard in all

the arteries by compressing them with the stethoscope (page 158); and at the same time the dilatation of the vessels may sometimes be felt strong enough to raise the observer's head. Hence the elongation of the aorta just alluded to: the other arteries are affected in a similar manner.

CHAPTER XXIV.

DILATATION OF THE HEART.

THE signs of dilatation of the heart are a bulging of the præcordial region, and an increase in the area of cardiac dulness to percussion. The præcordial bulging has been already described (page 33). The dulness to percussion is increased in the horizontal axis of the heart; to the right when the right cavities are involved, and to the left when the left side is dilated. But the shape of the dull space is not materially changed hereby; it remains oval; a point of value in the diagnosis between an enlarged heart and a pericardial effusion. Another important diagnostic sign of dilatation is that the place of impulse moves with the extension of dulness; both sometimes reach to the left as far as the axillary line. The enlarged heart tends to displace the lungs, and so to come into contact with a larger space of the chest-wall; hence an impulse more extensive than natural.

The sounds of a dilated heart are weak. The

Fig. 22.

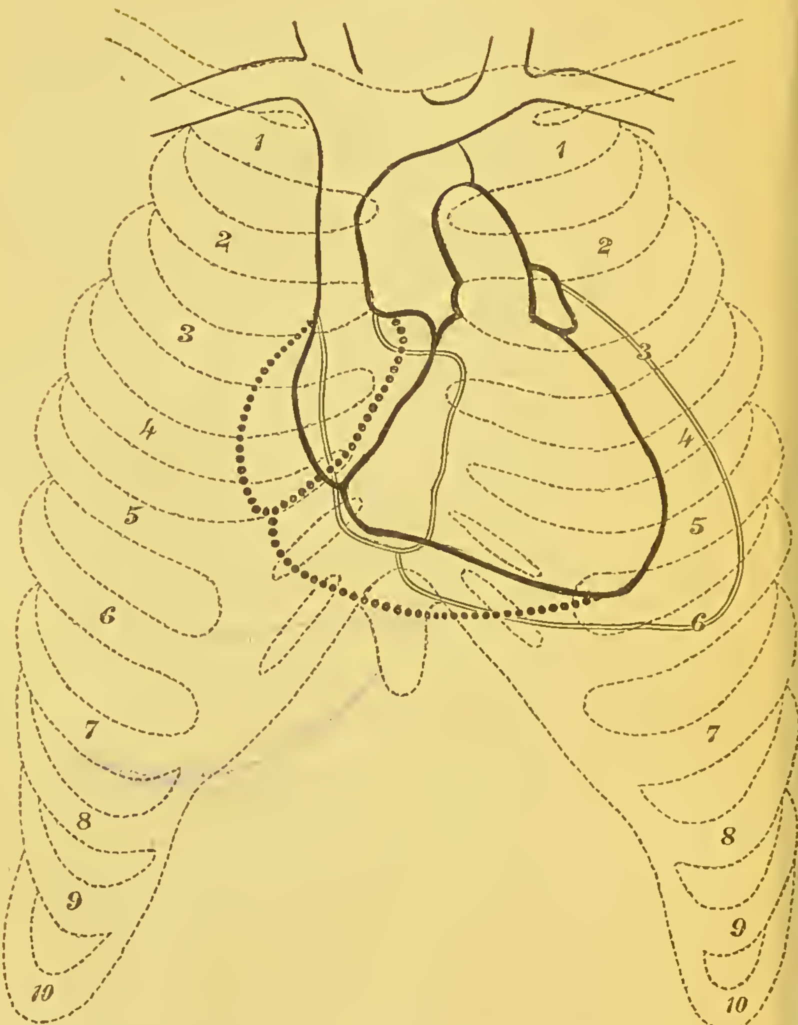


DIAGRAM SHOWING DILATATION OF THE RIGHT SIDE AND OF THE LEFT SIDE OF THE HEART: FROM VON DUSCH.

diaphragm is depressed, sometimes to such a degree that a liver of natural size may be deemed greatly enlarged. Dilatation of the right cavities tends to be accompanied by all the symptoms of asystolism (page 237) and obstructed venous circulation (page 162). Marked dulness to percussion and enfeebling of the respiratory sound, over the upper part or even the whole of the left lung, are not unfrequently present when the dilatation of the heart is great.[1]

[1] Compare the effect of pericardial effusion: page 247.

CHAPTER XXV.

PERICARDITIS.

THE physical signs of pericarditis depend upon the inflammatory effusion into the pericardium. When the quantity of the exudation is small, it cannot be discovered, unless it cause a friction-sound (page 152). For this reason, a friction-sound is usually the earliest sign afforded by pericarditis. When the quantity of the exudation is large, it is discovered by means of percussion.[1] Disappearance of a friction-sound is due either to progressing liquid effusion, or to adhesion of the inflamed surfaces. But friction is not always abolished even by a large effusion.

[1] See the next chapter.

CHAPTER XXVI.

PERICARDIAL EFFUSION.

INCREASE in the area of præcordial dulness; this is the sign, not only of an enlarged heart, but also of a liquid effusion into the pericardium. The two diseases however do not extend the area of dulness in the same manner; hence the means of diagnosis between them. I have already spoken of enlargement of the heart: it remains to describe the signs of a progressive pericardial effusion.

The first extension of percussion-dulness occurs at the base of the heart, where the great vessels enter, and where the pericardium hangs loosely round them, and is most distensible. At the beginning, the dulness is increased chiefly upwards, so as to reach the second rib, in the left parasternal line. A larger effusion will cause the dulness at the base to extend transversely also, so as to reach from the right side-sternal line to the left nipple line, and as high, it may be, as the first rib.

Hitherto there will have been little change in the signs afforded by that part of the pericardium which is close upon the diaphragm. However, further increase in the fluid, after it has distended the pericardial sac around the vessels, will dilate the pericardial sac around the heart. Hence, progressive increase in the transverse præcordial dulness below the base of the heart, and corresponding displacement of the lungs, until, in an extreme effusion, the non-resonant space will reach from the right nipple line to the left axillary line, and up to the top of the manubrium sterni. In which case, the area of dulness will obviously be triangular with the apex upwards. And in all pericardial effusions, whether great or small, it is the upward extension of dulness which affords the means of diagnosis from enlargement of the heart.

Gerhardt has found that, when the quantity of fluid is not very great, the dulness reaches higher in the upright than in the lying position of the body.[1] Partial pericardial adhesions, pleural adhesions, or emphysematous lung in front of the heart, will interfere with the regular development of dulness as just described.

[1] Lehrbuch, page 248.

When the effusion is limited to the base of the heart, the apex-beat and the left limit of dulness will correspond; but when the cardiac portion of the pericardium is distended, the dulness will reach proportionately beyond the apex-beat, and to the left of it: this is pathognomonic. At the same time the impulse is weakened; it may finally become imperceptible; in consequence of the separation of the heart from the front of the chest. And, inasmuch as the heart hangs free to take the lowest room in the dilated cavity, the position of the impulse shifts with the position of the patient. This sign Oppolzer regards as very characteristic:[1] but be it remembered that Gerhardt has proved that the heart, in a sound person, is moveable within very wide limits. Nevertheless, for an impulse wholly impalpable in the lying posture to become well felt in the erect, is a strong presumption in favour of a pericardial effusion. Præcordial bulging is consequent upon a large pericardial effusion as well as upon a dilated heart. In a few cases of excessive distension, a sort of undulation has been seen; comparable with pulsating empyema.

[1] Vorlesungen; herausgegeben von Stoffella. 1866, i. 13.

The lungs around a distended pericardium are relaxed. A large effusion sometimes exerts an amount of pressure upon the bronchi, especially of the left lung, such as to cause more or less complete collapse of the corresponding pulmonary lobe. And when this lobe happens to be the lower lobe, a pleural effusion is simulated. Nor can the evidences of pleural effusion, which the mediastinum and diaphragm usually afford, be appealed to, inasmuch as they are already under the influence of the pericardial effusion. Amid these circumstances, that sign of liquid in the pleura, upon which Damoiseau justly laid so much stress, will prove serviceable : the dulness and sense of resistance yielded by gentle percussion are much more intense over a liquid effusion than over collapsed lung. Graves once observed a great bulging of the left lung-apex above the clavicle, dependent upon pericardial effusion.

The diaphragm is depressed, and therewith the liver and the spleen. Occasionally the depression is so great as to produce a swelling of the epigastrium.[1] On the other hand, the diaphragm may be paralysed ; if it be, the epigastrium sinks inwards during inspiration.

[1] Inventum Novum, § 46.

The diagnosis is from enlargement of the heart; aneurysmal, or other intrathoracic tumours; suppuration of the mediastinum; consolidation of the lung in front: and pleural effusion.

The means of distinguishing the different kinds of pericardial effusion are the following. A friction sound, occurring at any time, points to an inflammatory effusion. When gas is present in the pericardial sac, the dulness gives way to a resonant note: the cracked-pot sound, and succussion splash, have been heard in such cases. But the liquid, which is always present along with the gas, yields a certain area of dulness which changes position along with change in the position of the body.

CHAPTER XXVII.

ADHERENT PERICARDIUM.

CONCRETION of the heart and pericardium does not admit of diagnosis, unless the pericardium have contracted external adhesions also with the walls of the chest in front and with the spinal column behind. Under these conditions of both internal and external past pericarditis, the following signs will be present. i. Systolic recession of the apex-beat (page 42): a sign which must be well-marked to be depended upon. ii. Deep inspiration does not diminish the area of superficial cardiac dulness: does not depress the apex-beat: and is attended by recession of the epigastrium, consequent upon the fixedness of the pericardial portion of the diaphragm. iii. Diastolic collapse of distended jugular veins (page 168). Internal and external pericardial adhesions necessitate dilatation and hypertrophy of the heart.

CHAPTER XXVIII.

MITRAL REGURGITATION.

A MURMUR, systolic, replacing or immediately following the first sound, heard louder at or just above the apex-beat than over any other part of the præcordia, conducted round the left side of the chest to the angle of the scapula and heard distinctly there, indicates regurgitation through the mitral orifice. In consequence of the reflux of blood, the left auricle and the vessels of the lungs are overfilled, the tension within the pulmonary artery is increased, and its sigmoids are closed with unnatural force. At the same time, the tension within the aorta is diminished, and its valve closes feebly. For these reasons, the second sound becomes, in mitral regurgitation, louder over the pulmonary artery than over the aorta. The intensified sound is sometimes, so to say, palpable. Dilatation of the right side of the heart is a further consequence of the embarrassed circulation through the lungs.

Wherefore, when the peculiar murmur is present, and attended by the two concomitants, intensified pulmonary second sound and dilatation of the right side of the heart; it is safe to diagnosticate mitral regurgitation.

Incompetence of the mitral cusps is not always attended by a murmur: this is especially the case in asystolism. Much more frequently, a systolic apex murmur is not due to mitral regurgitation. These non-regurgitant murmurs are, as a rule, inaudible behind at the angle of the scapula, and are unattended by intensification of the pulmonary second sound, or by enlargement of the heart. Dr. Andrew has shown[1] that, of these negative signs, the inaudibility behind is the most important: a murmur not heard at the angle of the scapula is rarely regurgitant. The lesser degrees of enlargement of the heart are not easily discovered. And the accentuation of the pulmonary second sound is a sign somewhat fallacious; not only for the reasons before given (page 188), but also because an obstruction to the circulation in the lung

[1] On certain Endocardial Murmurs. Saint Bartholomew's Hospital Reports, vol. i.-1865. See also: Austin Flint, On cardiac murmurs. Amer. Journ. Med. Sc. July, 1862.

itself will induce unnatural fulness of the pulmonary artery. The causes of these non-regurgitant murmurs are obscure. Roughness of the endocardium lining the ventricle; vegetations hanging into the cavity, but not impeding the closure of the valve; some change in the texture of the valves, such as to render them incapable of producing a tone; contractions of the heart so strong as to stretch the valves and tendinous chords unduly; local pressure upon the ventricle; all these have been suggested as possible causes.

Regurgitation of blood through the mitral orifice is sometimes due to temporary conditions, notably the relaxation of heart which occurs in profound debility, whether idiopathic (e. g., chlorosis, purpura)[1] or consequent upon acute diseases. The abiding causes of regurgitation have been before enumerated (page 151).[2]

[1] Dilatation of the heart in chlorosis has been noticed by many observers; Bamberger, Friedreich, Wunderlich, Vogel. See also, Stark: Vergrösserung des Herzens bei Chlorosis. (Arch. für Heilkunde, 1863). In this way regurgitation at the mitral or tricuspid orifices may be easily explained. The dilatation is due to debility of the muscular fibre, and is not necessarily permanent.

[2] According to Naunyn, a mitral regurgitant murmur is sometimes heard loudest over the pulmonary artery, in

consequence of the position of the left auricle, so close to the root of that vessel: the murmur produced at the mitral orifice passing backwards into the auricle. Berl. klin. Wochenschr. 1868, No. 17.

CHAPTER XXIX.

MITRAL OBSTRUCTION.

A MURMUR, after the second sound and before the first, heard louder at the apex-beat than over any other part of the præcordia, indicates obstruction at the mitral orifice. The murmur is usually præsystolic; because, although there is a passage of blood through the mitral orifice during the whole of the ventricular diastole, yet the current is greatly accelerated during the auricular systole. Inasmuch as mitral constriction is mostly associated with regurgitation, the præsystolic murmur commonly passes indistinguishably into a systolic murmur. Often enough, however, in spite of this complication, a first sound can be heard, produced by portions of the mitral valve still capable of vibration, or by the tricuspid valve alone. And, occasionally, there is a distinct interval between the diastolic murmur, and the first sound or systolic murmur, as the case may be. The præsystolic murmur is not often heard

at the angle of the scapula. A præsystolic apex thrill, running up into the impulse, is present in most cases of mitral obstruction: sometimes the thrill is diastolic.

Overfilling of the left auricle and of the pulmonary blood-vessels is carried to a high degree. Hence accentuation of the pulmonary second sound, and hypertrophy with dilatation of the right side of the heart. But dilatation of the tricuspid orifice and tension within the pulmonary artery are antagonistic, as the former progresses the latter diminishes, wherefore the loudness of the pulmonary second sound becomes gradually less. In like manner, the aortic second sound is enfeebled and finally disappears, on account of the small quantity of blood sent out into the aorta. For the same reason, atrophy of the left ventricle attends uncomplicated cases of mitral obstruction.

CHAPTER XXX.

AORTIC REGURGITATION.

A MURMUR, replacing the aortic second sound, and heard loudest at the second right interspace and along the sternum, indicates regurgitation through the aortic valve. The murmur is usually conveyed to the apex-beat. It is also conducted along the arteries; sometimes to a surprising distance: a sign which cannot however be trusted (except when the murmur is of peculiar musical quality) inasmuch as a diastolic sound may be produced in the arteries themselves, in cases of aortic regurgitation: nevertheless, a diastolic murmur, unquestionably seated at the mouth of the aorta, can sometimes be heard in the radial arteries. A systolic basic murmur also is commonly present: or at any rate the first sound is not clear. This systolic murmur is certainly not always due to obstruction at the aortic orifice, but is probably sometimes a result of the same condition which causes the intense arterial

murmurs, namely, the great tension of the walls of the arteries. A diastolic thrill at the second right interspace is occasionally perceptible.

Just as mitral diseases tend to be followed by enlargement of the right side of the heart, so do aortic diseases bring after them hypertrophy of the left ventricle. The left ventricle, during the diastole, becomes filled from the aorta as well as from the auricle, and consequently has, during the systole, an unnaturally large quantity of blood to expel. Hence the frequency of dilatation of the ascending aorta: hence the elongation of the arteries, and the visibility of the pulse: hence the hardness of the pulse,[1] and the intensity of the arterial systolic murmur: hence the phænomenon of a capillary pulse, observed by Lebert, that is to say, a systolic flushing of the face. Hence, lastly, the peculiar cardiographic sign which has been de-

[1] "Après avoir remarqué l'abattement de ses yeux, la bouffissure, et la pâleur de son visage, j'examinai son pouls qui me parut fort plein, fort vîte, dur, inégal, et si fort que l'artère de l'un et l'autre bras frappait le bout de mes doigts autant que l'auroit fait une corde fort tendue et violemment ébranlée." Histoire de Jean Chifort (case of disease of aortic sigmoids): Vieussens. Œuvres françoises, 1715.

scribed by Marey,[1] and which consists in a too rapid ascent of the traced line between the second sound and the auricular systole, indicative of a too rapid filling of the ventricle. Duroziez' diastolic arterial murmur sometimes becomes a valuable sign of aortic regurgitation (page 159).[2]

A mitral systolic murmur often concurs: due either to simultaneous implication of the aortic flap of the mitral valve, or to incompetency of the valve consequent upon dilatation of the ventricle, and relative shortening of the musculi papillares.

The diastolic murmur, the failure of the aortic second sound, the signs of hypertrophy of the ventricle, and the peculiar pulse, are the most important signs of aortic regurgitation. But numerous cases have been observed[3] of an

[1] Gaz. Méd. de Paris, 1868, No. 38.

[2] I wish to remark that Stoll has noted the coincidence of aortic regurgitation and a tendency to sudden death. In the sixteenth post-mortem examination described in Rat. Med., vol. i. p. 230, the aortic sigmoids were found so much diseased as to be quite incapable of closing the valvular orifice: "Mortem repentinam ab aucto humorum ad cor allapsu deduximus, qui ab osseo aortæ capite, valvulisque ferme ex toto rigidis expediri promoverique ulterius nequiverant."

[3] By Cruveilhier, Vulpian, Gubler, Fournier: see Edinb.

aortic diastolic murmur heard during life, not to be explained by any lesion of the aortic valve found after death. In all these cases, however, the aorta has been unnaturally rigid, if not dilated. Wherefore it seems that a murmur, in all respects like that of aortic regurgitation, may be produced immediately above the valves. Marey declares that the cardiograph affords the only means of diagnosis, the sign above described being quite peculiar to aortic regurgitation.

Med. Journ. vi. page 471. See also Marey: Gaz. Méd. de Paris, 1868, No. 38.

CHAPTER XXXI.

AORTIC OBSTRUCTION.

THE murmur present in cases of aortic obstruction is systolic, and heard loudest in the second right interspace. But many inorganic murmurs possess these characters. Wherefore it is not allowable to diagnose an obstruction unless the murmur be loud and long, and attended by active hypertrophy of the left ventricle, and by a pulse which is small even when the heart is beating strongly : moreover the signs of an aneurysm must be absent. A systolic thrill, felt on the right side of the base of the heart, is often present. Aortic obstruction is usually complicated with regurgitation : even when there is no diastolic murmur, the aortic second sound is muffled, weakened, or absent. The pulmonary second sound is unaffected, inasmuch as the mitral valve bears off the retrograde development of disease.

Inorganic aortic systolic murmurs are probably due to the walls of the aorta being thrown

into unusual vibration by the impact of the blood.[1] It is in states of debility, or during fits of palpitations, that inorganic systolic aortic murmurs are heard. They are always soft; and cease with the debility or the palpitations, as the case may be.

[1] Bondet believes that, in these cases, there is a real narrowing of the orifice, due to contraction of the elastic and muscular fibres contained in the valvular structures. Gaz. des Hôp. 1866, No. 135.

CHAPTER XXXII.

TRICUSPID REGURGITATION.

TRICUSPID regurgitation, in the great majority of cases, is merely relative, a part of dilatation of the right side of the heart, and secondary to impeded circulation through the lungs. The attendent murmur is systolic, and heard best over the lower part of the sternum. But commonly it is only when the distension of the orifice is extreme that the murmur becomes audible : for which reason, it is most frequently heard when the heart falls into a state of asystolism (page 237) : hence, moreover, the murmur is often temporary. The important signs of tricuspid regurgitation, which the jugular veins afford, have been already described (page 160). Weakness of the pulmonary second sound has been noticed once or twice, but the ordinary cause of tricuspid regurgitation (namely the obstructed pulmonary circulation) interferes with the occurrence of this sign. A systolic thrill at the epigastrium is said to have been present in

a few cases. Passive dilatation of the right heart is a constant accompaniment.

Mitral regurgitant murmurs may sometimes be mistaken for tricuspid : the diagnosis, in a difficult case, depends chiefly upon the fact that obstruction is often associated with regurgitation at the mitral orifice, but very rarely at the tricuspid. So that if a murmur be at all præsystolic it is probably mitral. Moreover a tricuspid murmur is usually very soft in character.

Parrot has sought to show that the so-called anæmic murmurs, which have been commonly regarded as produced in the pulmonary artery or in the aorta, are really due to tricuspid regurgitation, consequent upon relaxation of the heart's tissues.[1] He maintains that these murmurs are heard loudest at the left side of the sternum, in the fourth interspace (sometimes third or fifth) and that they are inaudible higher up. Certainly this is the seat of tricuspid murmurs, but I cannot agree that it is the ordinary seat of anæmic murmurs. However, it is not only possible but probable that a

[1] Etude clinique sur le siége et le mécanisme des murmures cardiaques dits anémiques. Arch. Gén. de Méd. 1866.

tricuspid regurgitation, like a mitral regurgitation may sometimes be the result of great debility.[1]

[1] A young man, twenty-four years old, came on Dec. 20 to the out-patient room of St. Bartholomew's Hospital. He said that he was quite well until Dec. 16, when, as he was getting up at four in the morning to go to his work, he felt, all at once, pain across his back below the tenth rib, and shortness of breath. He had no cough: had never spat up blood, or been liable to palpitations. Stooping caused his heart to beat strongly, and then a distinct systolic murmur was heard, almost localised to the sternal end of the fourth left intercostal space; inaudible at base or apex. As his heart became more quiet, the murmur ceased. To hold his breath did not bring on the murmur. Jugular veins almost invisible: lips distinctly livid: nothing amiss discoverable in lungs. When seen a week afterwards his condition was just the same.

CHAPTER XXXIII.

TRICUSPID OBSTRUCTION.

THIS is a rare disease. The murmur is diastolic, and heard loudest about the bottom of the sternum. In a few cases the murmur has been distinctly præsystolic, and accompanied by a præsystolic thrill.[1] The systemic venous circulation is obstructed. On the other hand, in simple tricuspid constriction, the tension within the pulmonary vessels would be reduced, and thereby the pulmonary second sound be weakened, were it not that mitral disease usually complicates the tricuspid.

[1] Gairdner: Edin. Med. Journ. vol. v. page 871. Haldane: Edin. Med. Journ. vol. x. page 271. Duroziez declares that tricuspid obstruction is accompanied by a systolic murmur. Gaz. des Hôp. 1868.

CHAPTER XXXIV.

PULMONARY REGURGITATION.

THIS, the rarest of all valvular diseases, is attended by a diastolic murmur, heard loudest in the second left interspace and along the sternum. The pulmonary second sound disappears, or is greatly altered. A diastolic thrill is sometimes present. A systolic murmur, not due to obstruction, occurs in pulmonary regurgitation like as in the corresponding aortic disease. Active hypertrophy of the right ventricle, and impeded systemic venous circulation, are secondary consequences.

CHAPTER XXXV.

PULMONARY OBSTRUCTION.

THE murmur of pulmonary obstruction is systolic, and heard loudest on the third rib, or in the second or third left interspace, close to the sternum.[1] A strong systolic thrill may sometimes be felt at the same spot. Active hypertrophy of the right ventricle supervenes; so that it is not until the obstruction becomes great, or the heart's contractions begin to fail in vigour, that there is any stagnation in the general venous system.

Inorganic murmurs frequently assume the time and place indicative of pulmonary obstruction. Sometimes they can be explained by the pressure of solidified lung (phthisical or pneumonic) upon the artery: sometimes pressure by the stethoscope produces the murmur, espe-

[1] Ormerod: On a systolic murmur in the Pulmonary Artery. Edin. Med. and Surg. Journ. 1846. Meynet: Rétrécissement de l'orifice de l'artère pulmonaire, consécutif à une endocardite valvulaire. Gaz. Méd. de Lyon. 1867.

cially in patients with a phthinoid chest: but often enough the murmur cannot be thus explained. When this is the case we may suppose that the impact of the blood against the arterial walls throws them into vibration sufficient to generate a sound. It is especially in states of debility and impoverished blood that pulmonary inorganic murmurs occur.

CHAPTER XXXVI.

INORGANIC MURMURS.

FOR the purposes of diagnosis I have spoken of these murmurs with reference to each form of organic valvular disease which they simulate. I recur to them here merely to remark that they are nearly always systolic, and are more common on the right side of the heart than on the left. Fatty degeneration of the cardiac muscular fibre, especially partial disease of the columnæ carneae; the granular degeneration consequent upon febrile, putrid, or cachectic states; and temporary coagulations, are frequent causes of 'inorganic' murmurs seated at the mitral or tricuspid valve. Pulmonary and aortic murmurs are mostly due to pressure, or to impact of the blood upon a vessel which has lost its tone.

CHAPTER XXXVII.

INFLAMMATION OF THE MEDIASTINUM.

THERE are different kinds of inflammation of the mediastinum: to wit:

i. Acute dropsy of the mediastinum. The connective tissue in all parts of the body is liable to acute serous fluxions, which are idiopathic in the sense of inexplicable. Spontaneous serous fluxion upon the mediastinum is a rare disease. Hamburger has narrated a case which probably was of this kind.[1] In this patient the posterior mediastinum chiefly was affected. There were present all the symptoms of acute obstruction to the vena cava superior. The heart was pushed forward, and beat strongly against the chest wall. The percussion signs were natural. When the patient swallowed some water, and, at the same time, the ear was applied to his back on the left side of the third

[1] Die Auscultation des Œsophagus. Vierteljahrschrift: Bd. i. Prag, 1870. Hamburger refers to a case described by Rayer.

dorsal vertebra and below; instead of a clear clucking sound, a weak obscure murmur was heard; probably indicative of enfeebled œsophageal contraction, and also of bad conduction of the sound which was produced. Hydromediastinum is usually associated with hydrothorax or hydropericardium as part of general dropsy.

ii. Adhesive inflammation of the mediastinum is chronic, and secondary to pleurisy (especially empyema) or to severe pericarditis. The only signs are those which are dependent upon the fixity of the heart, and these have been already described under the head of adherent pericardium (page 252).

iii. Suppurative mediastinitis also is usually a chronic disease. A collection of pus in the anterior mediastinum causes a corresponding extent of percussion-dulness. Even when the abscess comes to the surface the diagnosis is not always easy, for the tumour may pulsate so as to simulate an aneurysm. The diagnosis of posterior mediastinitis is not much assisted by physical examination. Auscultation of the œsophagus might prove serviceable, but has not been, so far as I know, actually practised.

CHAPTER XXXVIII.

MEDIASTINAL TUMOURS:

INCLUDING tumours developed in the connective tissue, and enlargement of the thymus, or of the bronchial glands. A large swelling sometimes causes protuberance of the front of the chest in the neighbourhood of the sternum: and this protuberance may be influenced by the pulsations of the aorta. The heart may be displaced downwards, or pressed forwards. Dulness to percussion will exist over a tumour which is in contact with the chest-wall: except that small tumours, intimately connected with the trachæa or a bronchus, will afford a clear, high-pitched note. The tumour conducts sounds generated in its vicinity or not: bronchial breathing, bronchophony, and the heart's sounds. Sometimes a systolic murmur at the base of the heart is produced by the pressure of the tumour. The œsophagus should be auscultated when disease in the posterior mediastinum is suspected. Gangrene of one lung

often ensues when its root is compressed by a mediastinal tumour. The signs of this kind of pulmonary gangrene are dulness to percussion, and great weakening or complete absence of respiratory sound. Sometimes the gangrenous matter is expectorated; whence all the signs of cavity: this event often occurs in the upper lobe of the lung.

CHAPTER XXXIX.

ANEURYSM OF THE THORACIC AORTA.

ANEURYSMS of the thoracic aorta must have attained a size sufficient to bring them into contact with the chest-wall before they yield any physical signs directly dependent upon the aneurysm itself. Earliest to appear are the signs afforded by palpation and percussion.

By palpation are detected pulsations and thrills. The pulsation is systolic, synchronous with the impulse of the heart. A slight diastolic shock, due to forcible closure of the aortic sigmoids, is sometimes perceptible over an aneurysmal tumour : in a case seen by Dr. Walshe there was a strong diastolic impulse.[1] When the aneurysm contains much coagulum, the pulsation is weak or even absent. Aneurysm of the ascending aorta touches the chest-wall first in the second right interspace close to the sternum :

[1] Diseases of Heart. 1862, page 457.

along with increase in the size of the tumour, the pulsations become felt upwards towards the manubrium sterni, or downwards, along the right margin of the sternum, haply as low as the fourth interspace.[1] Aneurysm of the transverse aorta first comes to the surface behind the upper part of the sternum, and afterwards extends far away towards the left.[2] Aneurysm of the descending aorta, at its upper part, points below the first left rib, and thence extends downwards to the second space. Aneurysms of the lower part of the thoracic aorta lie upon the left side of the dorsal vertebræ and cause pulsation there. The site of pulsation sometimes is also the site of systolic or diastolic thrill.

By percussion, dulness may be discoverable in the same situations : namely, in the neighbourhood of the second and third ribs along the

[1] An aneurysm of the innominate artery lies behind the first rib close to the sternum : the left innominate vein is compressed early in the disease ; when the tumour becomes large, the right vein also may be involved.

[2] Aneurysm of the pulmonary artery, an extremely rare disease, sometimes produces a pulsating tumour seated at the sternal end of the second and third left intercostal spaces : the diagnosis from an aortic aneurysm depends upon the absence of dulness, pulsation, or other sign of tumour beneath the manubrium sterni.

right side of the sternum; under the manubrium sterni; to the left side of the sternum; and along the left side of the dorsal vertebræ. The aneurysmal dulness is sometimes conterminous with the cardiac dulness, sometimes not.

By inspection, a tumour upon the surface of the chest is sometimes detected. The position of the tumour corresponds with that of the palpation and percussion signs. The shape of the tumour is hemispherical, except that it is sometimes grooved where it is constricted by resistent fibres in the overlying parts. An aneurysmal tumour of the descending aorta may be of a size so great as to push the scapula outwards.

By auscultation there will be discovered a first and second sound, or a systolic and diastolic murmur, or a first sound and diastolic murmur, or a systolic murmur and a second sound, or no sound at all. The first sound is probably produced by rhythmical vibration of the aneurysmal walls; the second sound is the second sound of the heart conducted; the systolic murmur is due either to irregular vibration of the walls of the sac, or to the passage of blood through a narrow mouth into the aneurysm; the diastolic murmur is either a conducted murmur produced

at the mouth of the aorta, or is due to the passage of blood out of the sac, or is inexplicable.

Simple uniform dilatation of the ascending aorta is attended by extension of percussion dulness to the right of the margin of the sternum, and on the level of the second and third cartilages. Simple elongation of the aorta forces the base of the heart downwards and to the left; in which case the apex-beat comes to be lower and more external than natural.

An aneurysmal tumour, like other mediastinal tumours, may compress neighbouring viscera. Pressure upon the spongy structure of the lung produces relaxation or collapse of the part involved: pressure upon the root of the lung tends to produce pulmonary gangrene: pressure upon the œsophagus probably produces signs like those which were present in Hamburger's case of mediastinitis (page 274); I am sorry not to have had the opportunity of ascertaining whether this be so or not. The heart is liable to sundry displacements, thus: dilatation of the ascending aorta usually implies elongation thereof, whereby the heart is forced downwards and to the left: a large tumour of the transverse aorta produces the same effect: a very large tumour of the descending aorta will

push the heart towards the right: a tumour behind the heart will press it against the front wall of the chest.

There are upon record a few cases in which an aortic aneurysm has broken into the upper vena cava. If the opening into the vein be large and free, the dilated jugular veins will pulsate.

SUPPLEMENTARY NOTE UPON VOCAL RESONANCE.

By universal custom, whispered sounds, heard over the surface of the chest, have been considered to belong to the topic of vocal resonance. I am now convinced that this is a mistake. The kind of speech, which we call whispering, is in no way due to vibration of the vocal chords ; and is nothing more than a loud expiratory sound, produced by the passage of air through the inactive rima glottidis, and modified by the cavities above the larynx in the same manner as the tones of the voice (that is of the vocal chords) are modified. Whispering is not a vocal sound at all, in the true sense of the word vocal ; and the theory of whispered sounds, as heard by auscultation, is in fact the same as the theory of respiratory glottidean sounds (page 119).

INDEX.

THE END.

BRADBURY, EVANS, AND CO., PRINTERS, WHITEFRIARS.

ILLUSTRATIONS OF DISSECTIONS, in a Series of Original Coloured Plates, the size of life, representing the Dissection of the Human Body. By G. V. ELLIS, Professor of Anatomy in University College, London, and G. H. FORD, Esq. Complete in 29 Parts, Imperial folio, price £5 3*s.*, or half-bound in Morocco, price £6 6*s.*

Parts I. to XXVIII., each 3*s.* 6*d.* Part XXIX. 5*s.*

"With these plates, and such as these, by his side, the learner will be well guided in his dissection; and under their guidance he may safely continue his study when out of the dissecting-room.—With such plates as these, the surgeon will be fully reminded of all that is needful in anatomy when engaged in planning an operation."—*Medical Times.*

DR. MURPHY'S PRINCIPLES AND PRACTICE of MIDWIFERY. Second and greatly Improved Edition. With many Illustrations. Complete in 1 vol., small 8vo. 12*s.* 6*d.*

"In discussing the treatment, Dr. Murphy is not content with laying down arbitrary rules, but refers constantly to the principles on which he acts: while his directions are clear, explicit, sound, and eminently practical."—*Dublin Quarterly Journal of Medical Science.*

DR. ROBERTS ON URINARY AND RENAL DISEASES. A Practical Treatise. Illustrated by numerous Cases and Engravings. By WILLIAM ROBERTS, M.D., Physician to the Royal Infirmary, Manchester. Second Edition, thoroughly revised and much improved. Small 8vo. (*Preparing.*)

"We commend the work most cordially as admirably adapted to supply in medical literature a treatise which every intelligent physician must have felt to be needed."—*American Journal of Medical Science.*

DR. GEORGE HARLEY ON DIABETES: ITS Various Forms and Different Treatments. Small 8vo. 2*s.* 6*d.*

DR. GEORGE HARLEY ON ALBUMINURIA, WITH OR WITHOUT DROPSY: its Different Forms, Pathology and Treatment. Small 8vo. 2*s.* 6*d.*

QUAIN AND WILSON'S ANATOMICAL PLATES. 201 Plates. Royal Folio. 2 vols., half-bound morocco, £8 8*s.* Coloured.

*** *Sold also in Divisions (separately) as under—*

			PLAIN.	COLOURED.
MUSCLES	(51 Plates)	Cloth	£1 5 0	£2 4 0
VESSELS	(50 Plates)	,,	1 5 0	2 0 0
NERVES	(38 Plates)	,,	1 1 0	1 14 0
VISCERA	(32 Plates)	,,	0 17 0	1 10 0
BONES & LIGAMENTS	(30 Plates)	,,	0 17 0	1 0 0

DR. GARROD ON GOUT AMD RHEUMATIC GOUT. Second Edition, with extensive alterations. Coloured and other Illustrations. Small 8vo. 15*s.*

"Dr. Garrod has in this edition incorporated the results of his increased experience of the nature and treatment of gout; and has added a chapter on the diseases to which gouty persons are peculiarly liable."—*British Medical Journal.*

DR. WALSHE ON DISEASES OF THE HEART AND GREAT VESSELS, including the Principles of Physical Diagnosis. Third Edition. Revised and greatly Enlarged. Small 8vo. 12*s.* 6*d.*

DR. WALSHE ON DISEASES OF THE LUNGS, including the Principles of Physical Diagnosis. Third Edition. Revised and greatly Enlarged. Small 8vo. 12*s.* 6*d.*

HENRY'S GLOSSARY OF SCIENTIFIC TERMS for GENERAL USE. 12mo. 3*s.* 6*d.*

CHEMISTRY FOR SCHOOLS. By C. H. GILL, late Assistant Examiner in Chemistry in the University of London. Many Illustrations. Small 8vo. 4*s.* 6*d.*

"Mr. Gill has fairly earned the thanks of scientific chemists; nor will the schools be slow to appreciate a manual which has been thus well-devised and executed by an author who has himself been a successful school teacher."—*Nature.*

"As a guide in chemistry for boys of the higher classes, the book is extremely good. Those, especially, who are preparing for the matriculation examination of the University of London will find here just what they want."—*British Medical Journal.*

DR. HOFMANN'S MODERN CHEMISTRY, EXPERIMENTAL AND THEORETIC. Many Illustrations. Small 8vo. 4*s.* 6*d.*

"It is in the truest sense an introduction to chemistry; and as such it possesses the highest value,—a value which is equally great to the student, new to the science, and to the lecturer who has spent years in teaching it."—*Reader.*